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THE CRICO-ARYTENOID JOINT.

A Diarthrodial Articulation Subject to Rheumatoid Arthritic Involvement.*†

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In 1952 the author's attention was first drawn to a clinical situation which was unique to him. At that time, and until December, 1955, he was not aware of this clinical entity having been described in the American otorhinolaryngological literature.

Since 1952 he has seen a total of six cases, and since December, 1955, when Montgomery, Perone and Schall[29] wrote on the subject, two other articles have appeared in the American literature[37,40]; and in all probability there will be several more in the near future.

The purpose of this endeavor, therefore, is not to belabor the subject further, but to make the following points:

1. That what is "new" to one, may well be "old hat" to others, particularly where the others are intellectually more broadened and/or intelligent enough to recall, or re-explore, basic texts.

2. That there is really very little "new," if one takes the trouble to search the old literature, particularly in our special-

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ty, that of the Germans and English, during the latter half of the last century and the first decade of this century.

3. That it cannot help but stir up a great sense of admiration and respect for some of our forebears in otorhinolaryngology when one sees their remarkably astute observations and clinical acumen at a time when their armamentarium must have been exceedingly crude compared to currently available aids in diagnosis.

4. That, by the same token, there is much in the literature which appears to be present as a matter of expediency. Certainly there is considerable evidence to support the belief that in many institutions of higher learning today men labor mightily to produce mice. It would seem that they must "publish or perish." This is most regrettable. I hasten to add that this deplorable state is not by any means limited to medicine.

Further, the author would like:

5. To review the anatomy, gross and histologic, of the crico-arytenoid joint, and its place in the classification of joints.

6. To present evidence that this joint may become deranged, and lastly:

7. To indicate that we must, if we are to be good otolaryngologists, first be good physicians; conversant with the current thinking not only of our colleagues in general surgery (I have in mind the new 1960 certification requirements by the American Board of Otolaryngology), but also of our friends in internal medicine.

NARRATIVE CASE REPORT.

I feel it is of sufficient interest and importance to the purpose of this paper to relate my initial experiences with this clinical syndrome—bilateral mid-line fixation of the arytenoids and vocal cords—in narrative form, in the hope that anyone confronted with it may recognize it early and treat it properly. I say this because *1.* even after a diagnosis of *fixation* was established, this case was referred to as a "bilateral abductor paralysis" on numerous occasions, by house and attending

staff members, and *2.* because a number of laryngologists, pathologists and internists of considerable experience with whom this case was discussed, were not fimiliar with the syndrome.

Hastily, I confess that my search of the literature *at that time* was far from intensive, and by December, 1955, when the article on "Arthritis of the Crico-arytenoid Joint" by Montgomery, et al.,[29] was published, I had personally seen four cases; so it was with some satisfaction that I noted in their opening paragraph that they ". . . were unable to find a single American article on this subject in a review of the literature of the past thirty years." This feeling of warmth was preceded by the chilling news from the same authors that "arthritis of the crico-arytenoid joint is described in American otolaryngological textbooks;" and so it is, in the texts by Boies,[4] Morrison,[30] and Thomson and Negus,[47] as well as a few others.[3,21,28,42]

Rather than present all six cases in the usual style, I take the liberty of relating, in narrative form, my experiences with the first case I encountered, interspersing history and physical findings with the thinking (and lack of it) which transpired. The other case histories will be reported in the usual, more brief form.

Case 1. G.M., a 57-year-old white male, was admitted to the Brooklyn Veterans Administration Hospital on Nov. 17, 1952, with a chief complaint of difficulty in breathing of one week's duration. This had been getting progressively worse up to the time of admission. For several weeks prior to this, there had been slight hoarseness. There was no pain and no dysphagia. There had been no weight loss and no fever.

His past history revealed occasional episodes of "asthma" of about ten years' duration for which he had been treated irregularly and at widely separated intervals. Included in his past history was an admission to another hospital eight months before. The only available clinical record from that institution indicated that he had been admitted there on March 27, 1952, because of "difficulty in respiration" following an upper respiratory infection. Apparently direct laryngoscopy was performed, the details of which were not included in their resume. There was no indication that this had been preceded by indirect laryngoscopy, though in all probability it had been done, nor were findings on this procedure noted; however, their report did state that "pathological microscopic examination (of tissue presumably removed at laryngoscopy) was reported as "negative for malignant cells." They also reported that "sputum was negative for acid-fast bacilli." The patient was discharged on April 26, 1952, for follow-up at their clinic. (In the light of my own subsequent experience, this previous hospital admission is a very revealing and important part of his past history and will be dwelt on subsequently).

Of further importance in his past history was the absence of any diseases suggestive of diphtheria or typhoid fever. There was no history of trauma to the neck and no history of thyroid or other anterior neck surgery. The patient denied having ever had any venereal disease, and there was no history of joint pains or arthritis.

Physical examination on admission revealed a well-developed, well-nourished white male with moderate inspiratory stridor, moderate apprehension, but no cyanosis. The voice was only slightly hoarse. Blood pressure 146/84. Temperature 98.8° F. Pulse 88. Auscultation revealed wheezes throughout the chest but no moist or dry rales. Remainder of physical findings were negative except for indirect laryngoscopy, which revealed a slit-like glottic chink, with the vocal cords "being held in adduction" by a flat adhesive band running across the rima glottidis from the mid-portion of the free edge of one cord to the free edge of the other. This band of tissue was about 2 to 3 mm. in width anteroposteriorly.

With these findings on mirror laryngoscopy it was easy to reason that a traumatic direct laryngoscopy had been performed at the time of his earlier hospital admission, resulting in denudation of both cords with subsequent formation of a synechia or adhesion and his present resultant respiratory difficulty. One wondered what tissue had been biopsied or removed, and why, since (as noted) the report from that hospital merely stated "negative for malignant cells." One readily visualized a heavy-handed, not-quite-necessary laryngoscopy by some unidentifiable inexperienced colleague—wisely kept these thoughts to himself—and made plans to "fix the patient up in short order."

Meanwhile, the following laboratory and X-ray data came through: Complete blood count was normal. Serological test for syphilis was negative. Urinalysis normal. Chest X-ray revealed no evidence of cardiovascular, pulmonary, pleural or mediastinal pathology.

Approximately 36 hours after admission the patient was taken to the operating room for direct laryngoscopy for lysis of the laryngeal synechia. The Jackson anterior commissure laryngoscope was introduced with the beveled tip in the anterior commissure, bowing both cords outward anterior to the synechia (which was in the mid-portion of the middle third of the cords), and putting this adhesive band on the stretch. With a laryngeal knife the synechia was severed without difficulty.

Lo and behold! Both vocal cords remained fixed in the midline with a glottic chink at the posterior commissure of not more than 2 mm. The scope was gently but firmly introduced a bit further in an effort to abduct the cords, but these were completely resistant, being bowed outward anteriorly but with no motion attainable of either arytenoid. Further efforts to abduct the arytenoids with a closed laryngeal forceps were of no avail. (We were performing the "passive mobility test" of C. Jackson[21] without knowing it, v. i.). As a result of this turn of events, direct laryngoscopy was immediately followed by tracheostomy to assure an adequate airway.

It was now evident that we were dealing with a case of bilateral crico-arytenoid fixation . . . and what other morbid process besides an "arthritis" could be responsible. In order to have an arthritis, there had to be a joint; and while I had many times before used the term "crico-arytenoid joint," I had never thought of this area as a true joint.

It was also evident that in all probability this patient's crico-arytenoid joint had been involved in a slowly progressive pathologic process of a low-grade inflammatory or degenerative nature, possibly of many years' duration. It was felt that it was quite conceivable that this condition existed at the time of his prior hospital admission eight months before

because of "difficulty with respiration." It was also reasonable to assume that his synechia would never have developed, isolated in the middle of the cords as it was (rather than in the form of an anterior webbing) were it not for the fact that even then his cords were relatively immobile *in adduction*; furthermore, harking even farther back, it was my feeling that in all probability this patient's "asthma" of ten years' duration may have been, rather, an inadequate glottis. (One recalls the aphorism of Chevalier Jackson, "All is not asthma that wheezes.") These feelings were borne out by several things: *1.* that in going over his past history it became evident that most of his "asthmatic attacks" were almost always concurrent with upper respiratory infections; *2.* that there had been little or no relief from the usual anti-asthmatic drugs; *3.* the fact that during his current hospital admission a thorough allergy work-up revealed no evidence of positive reactions to any significant allergen. The Allergy Service stated that they felt he was "a non-reactive asthmatic;" *4.* that from the time of his tracheostomy, and all through his subsequent course in the hospital, for a period of almost seven months, he had no further "asthmatic attacks."

It was suggested to the patient that he have an arytenoidectomy so that he might be rid of the tracheotomy tube. It took him a number of months to agree to this. During this time the patient was put on a course of Cortisone therapy, and the larynx was re-examined periodically. No motion of vocal cord or arytenoid was seen, and, consequently, it was not until June 9, 1953, that surgery was performed. On that date a right arytenoidectomy was done via a laryngofissure approach according to the method described by DeBord.[11] His postoperative course was uneventful. Incidentally, fortunately the previous synechia had not reformed, and the cord edges were smooth. On July 20, 1953, the glottic chink measured about 5 mm. at the posterior commissure with the right cord about midway between complete abduction and adduction. On phonation the left arytenoid and cord adducted perhaps a millimeter or two, but did not abduct at all. Consequently the airway was adequate, and there was a quite serviceable, slightly breathy voice. The patient was decannulated. He has had no known difficulty since.

This is what was learned: Given a patient with dyspnea in whom the laryngeal mirror reveals a bilateral mid-line position of the vocal cords, the diagnosis of abductor paralysis is not justified, *a.* if there is no history of neck trauma; *b.* if there has been no thyroid or other low anterior neck surgery; *c.* if there is no chest lesion (even though it would be difficult to see how a chest lesion could involve both right and left recurrent nerves without other symptoms or signs); *d.* if there is no history of diphtheria or typhoid fever; *e.* if there is no history or serologic evidence of syphilis; *f.* if there is no evidence of other cranial nerve involvement besides the vagus, since it is hardly possible for any kind of central lesion to involve the nuclei or tract of the Xth nerve and no other.

These considerations alone should be sufficient to make one practically certain that there is fixation of the crico-arytenoid joint, rather than a bilateral abductor paralysis.

The final diagnosis can be unequivocally established by making, via direct laryngoscopy, the "passive mobility test" described by C. Jackson.[21] In paralysis the cords are flaccid and are easily abducted passively. In fixation, the anterior two-thirds may be forcibly bowed slightly; but the vocal processes remain close together and parallel to one another, and the arytenoid prominence is relatively immobile.

ANATOMY.

Unfortunately, not all standard texts on human anatomy describe the crico-arytenoid joint *per se*. In describing the cricoid and arytenoid cartilages, their articular facets are mentioned; however, Todd,[48] in the section titled "Articulations, Ligaments and Membranes of the Larynx" gives the following description:

"Crico-arytenoid Joints: Each of these joints has a ligamentous capsule lined by a synovial layer. The cricoid articular surface is convex, whereas that of the arytenoid is concave; both are elliptical in form and they are applied to each other so that the long axis of the one intersects or crosses that of the other at an acute angle. In no position of the joint do the two surfaces accurately coincide—a portion of the cricoid facet is always left uncovered. The capsule of the joint is strengthened behind by a band, which is inserted into the postero-medial part of the base of the arytenoid cartilage and plays an important part in the mechanism of the joint: it effectually arrests excessive forward movement of the arytenoid cartilage. The movements which take place at the crico-arytenoid joints are gliding and rotatory. During easy, quiet breathing, the arytenoid rests upon the lateral part of the cricoid facet. It can glide upon the cricoid facet and pass toward or from the medial plane and its fellow of the opposite side. In the rotatory movement the arytenoid cartilage revolves around a vertical axis; by this movement the vocal process is swung laterally or medially so as to open or close the rima glottidis."

The author goes on to describe a more or less rudimentary joint between arytenoid and corniculate cartilages, with a capsule but with no synovial lining.

Maximow and Bloom[28] have this to say about joints and synovial membranes:

"Bones are joined to one another by connective tissue structures which permit varying degrees of movement between the adjoining bones" (or, as in our subject, cartilages). "Such structures are called joints or articulations. These present extreme variations in character which depend primarily upon the type of bones which are joined and the varying degrees of motion permitted by the articulation. Thus, in some cases, as in the skull, the joints are immovable and the connected bones are separated by only a thin connective tissue layer, the sutural ligament.

Other joints are slightly movable such as the intervertebral articulations. Here the succeeding vertebrae are joined to one another by dense fibrous tissue and cartilage. Still other bones are freely movable upon one another, and here the bones are completely separated by cartilage and fibrous capsules."

Articulations in which there is little or no movement are called synarthroses. Of these there are three types: if the connection between the bones is of bone, it is called a synostosis; if of cartilage, a synchondrosis; and if of connective tissue, a syndesmosis. Joints which permit free movement of the bones (or cartilages) are called diarthroses.

We have thus established, it seems fair to state, that the crico-arytenoid joint is actually a true diarthrodial articulation. It is composed of two cartilages (which, incidentally, with advancing age, ossify in an orderly sequence, according to Pressman and Kelemen[36]) which move freely on each other; there is a definite joint capsule between them; each has an articular surface; each surface and the capsule is lined with synovial membrane; yet nowhere has the author seen this joint listed among diarthrodial articulations.

In diarthrodial joints there is a cavity. The walls of the joint cavities are composed of a dense connective tissue, whose cells are irregularly distributed and seldom suggest epithelium in arrangement. Occasionally small amounts of cartilage as well as all transitions between the cartilage cells and the joints or synovial cells can be found.

Articular surfaces are composed of, or covered with, hyaline cartilage. Where opposing cartilages touch they are not covered with dense connective tissue, but at their bases a small area of perichondrium is reflected backward into the membrane of the joint capsule. Articular cartilages, as is the case with most of the cartilage of the body, contain no blood vessels.

The synovial membrane lining the articular surfaces and capsule as a continuous unbroken lining may be thrown into either temporary folds which depend on the position of the joint, or they may form permanent villi which project into the joint cavity. There are no villi shown in any of the illustrations in this paper; however, Pearson[35] in a recent article

describes villi projecting into the joint cavity of an individual with reheumatoid arthritis of the crico-arytenoid joint. Generally there is an increase in the size and number of villi with age, and his patient was 67 years of age.

Many of the points of anatomical interest noted above may be seen in Figs. 1, 2 and 3. The crico-arytenoid joints of a

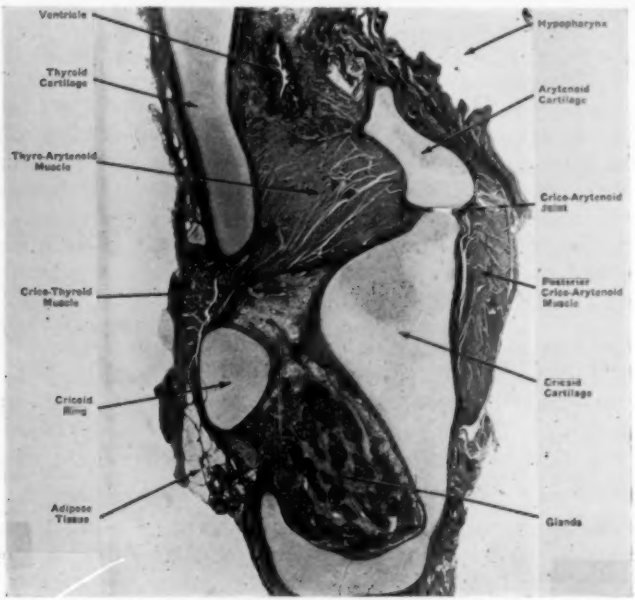

Fig. 1. Microphotograph (X7) of sagittal section of larynx of a nine-month-old infant showing the crico-arytenoid joint.

dog and a cat are shown for comparison in Figs. 4 and 5, respectively.

Having learned as a result of my first case (v.s.), to consider this a true joint, subject to arthritic changes and resultant fixation, I should like to have been able to report that my subsequent experiences with this syndrome were not so breathtaking (if I may be permitted this play on words) to

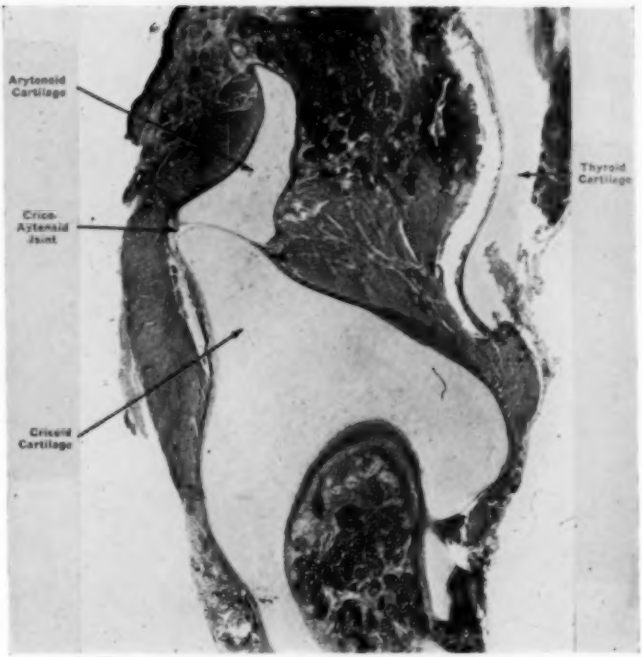

Fig. 2. Microphotograph (X9) of sagittal section of larynx of a nine-month-old infant showing the crico-arytenoid joint at about the mid-portion of the joint. Compare with Fig. 3, which is a similar section from same specimen, but about 1.5 cm. more medial.

either the patient or myself, but unfortunately, as will be seen from the following case reports, this was not universally true.

ADDITIONAL CASE REPORTS.

Case 2. J.D., a 58-year-old white male had been admitted to the Surgical Service of the Brooklyn Veterans Administration Hospital on Dec. 3, 1952, for excision of a ganglion of the right wrist. On admission it was noted that he was wearing a tracheotomy tube, through which he breathed, and which he expertly occluded with his finger when he wanted to speak. Otolaryngologic consultation was requested. After excision of the ganglion he was transferred to the Otolaryngology Service on Dec. 19, 1952.

Past History: Strenuous efforts to establish the reason for his tracheotomy resulted only in our finding that in July, 1945, at another hospital, he had had this tracheotomy performed because of increasing dyspnea

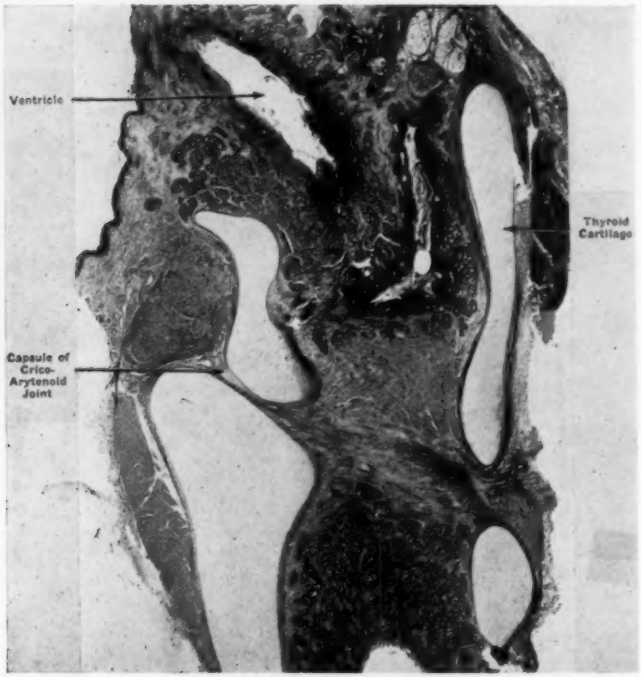

Fig. 3. Microphotograph (X9) of sagittal section of larynx of a nine-month-old infant. This section is about 1.5 mm. more medial than that in Fig. 2 and shows tangentially the medial portion of the joint capsule as compared to the slit-like joint space in Fig. 2.

over a period of about two months. Other than the tracheotomy he remembered no specific therapy and did not recall having been told exactly why he needed to wear this tube. Repeated efforts on our part to obtain hospital extracts or summaries were unsuccessful. It is noteworthy that for a period of seven-and-a-half years from July, 1945, to December, 1952, this patient worked as a taxicab driver in a bustling metropolis, and was apparently socially and economically adequate.

The remainder of his past history was non-contributory. There had been no diphtheria or tyhoid fever. He denied having had venereal disease. There was no history of trauma to the neck. There had been no thyroid or other anterior neck surgery. There was no history of arthritis or rheumatism.

Review of Systems was negative.

Physical Examination revealed a well-developed, well-nourished white male, lucid and cooperative. Blood pressure 116/78. Pulse and respiration normal. A No. 6 tracheotomy tube was in place. Indirect laryngo-

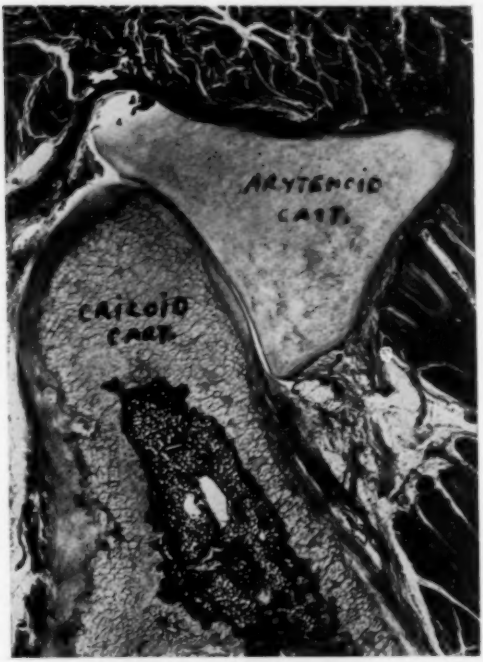

Fig. 4. Microphotograph (X14) of sagittal section of larynx of a dog through crico-arytenoid joint. Note marrow-like appearance within cricoid cartilage.

scopy revealed both arytenoids and vocal folds to be immobile in adduction, with a very narrow glottic chink. With the tube occluded there was marked inspiratory stridor but a rather good voice. The larynx otherwise appeared normal in all details. Neurological examination was entirely normal.

Laboratory and X-ray Data was as follows: Hemoglobin, 14.1 Gm.; r.b.c., 4.2 million; w.b.c. 9,700 with normal differential; blood urea nitrogen, 22.5. Blood serology negative. Urinalysis normal. Chest X-ray negative for cardio-vascular, parenchymal, pleural, or mediastinal pathology.

Course in Hospital: (Because Case 1 above, had been seen only one month before this time the presumptive diagnosis included crico-arytenoid joint ankylosis). On Dec. 21, 1952, direct laryngoscopy was performed under general inhalation anesthesia via tracheotomy tube, with complete muscle relaxation. Using the anterior commissure laryngoscope only the anterior two-thirds of the cords could be slightly bowed outward. The posterior one-third (occupied by the vocal processes) and both arytenoids resisted any lateral displacement whatever. Further efforts to move the

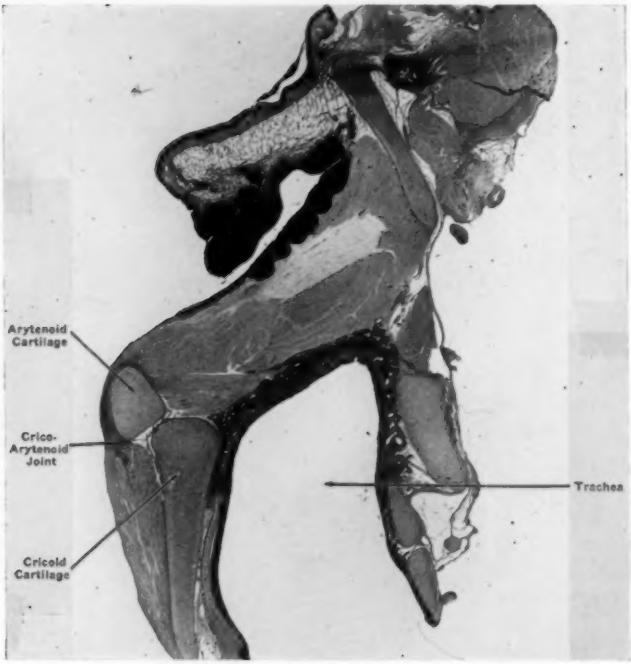

Fig. 5. Microphotograph (X8) of sagittal section of larynx of a cat through crico-arytenoid joint.

arytenoids and cords passively with a closed laryngeal forceps were also unsuccessful.

A diagnosis of bilateral crico-arytenoid joint fixation (presumably due to arthritis) was made.

The patient was discharged, to return for arytenoidectomy at a later date. He was most anxious to submit to this surgery in order to be rid of the tracheotomy tube.

He returned on Jan. 27, 1953. Careful examination for signs of other joint pathology was fruitless. The Rose test, a non-specific serologic complement-fixation test for arthritic activity was negative.

On Feb. 3, 1953, under general anesthesia, a right arytenoidectomy according to the method of Woodman[10] was performed. The postoperative course was uneventful. The patient was decannulated and was discharged on Feb. 17, 1953. The laryngeal airway was adequate, being about 5 mm. at the posterior commissure.

Because of the long-standing tracheotomy this tract was completely epithelialized and while its sides fell together, it did not heal closed;

therefore, he was subsequently re-admitted, and on Sept. 22, 1953, the tracheal fistula was excised and the wound closed in layers. This healed *per primum*, and the patient was discharged for the last time on Oct. 7, 1953. He has been seen at irregular intervals since then in follow-up. His airway is adequate and voice satisfactory.

Case 3. I.L., a 56-year-old female, was admitted as a private patient to the Long Island College Hospital on July 15, 1953, on the service of Dr. Benjamin Burbank, an Internist. Her *chief complaint* was "difficulty in breathing."

Present Illness: The patient was in good health until seven years prior to admission. Shortly after the onset of menopause she experienced episodes (she called them "attacks") of difficulty in breathing. These were manifested by both inspiratory and expiratory distress which was accompanied by a crowing voice. Similar "attacks" lasting from several minutes to several hours occurred intermittently, at more frequent intervals. It was also noted by the patient that over the past seven years even between attacks there was some increasing dyspnea on exertion.

Seven weeks prior to admission these episodes increased in duration and severity to become almost continuous. At that time she entered another hospital where she was told after several days that there was "no organic trouble." She had been seen by several physicians and had been on a number of medications, the exact nature of which were unknown. On occasion she had been given adrenalin for her attacks, and also a nebulizer. Neither of these resulted in any relief.

The nature of the crowing respiration (which sounded like the voluntary crowing one can accomplish by voluntarily inspiring with cords in light adduction—as opposed to the wheeze one would get in tight adduction, or laryngospasm), the type of apprehensive, nervous patient with whom we were dealing, the lack of response to anti-asthmatic therapy, and the lack of physical findings on chest examination, were responsible, I feel sure, for the diagnosis of "no organic trouble."

Review of Systems had been negative.

Past History was entirely non-contributory. There had been no typhoid fever and no diphtheria. The patient denied venereal disease. There had been no trauma to the neck and no thyroid surgery. There was no history of joint pains, arthritis or rheumatism.

Physical Examination on admission had revealed a well-developed, well-nourished white female in moderate respiratory distress, with crowing inspiratory noises which fluctuated in intensity, with no cyanosis. There was no dysphagia. Because of the dyspnea the patient spoke with frequent pauses between words or phrases, but the voice was relatively good, there being little, if any, hoarseness. Blood pressure was 120/90. Pulse 90 to 100. Respirations 24 to 30.

Laboratory Data: R.b.c., 4.63 million; Hgb., 14.0 gm.; w.b.c., 9,250 with normal differential. Sedimentation rate 11. Hematocrit 46 per cent. Fasting blood sugar 128. Urea nitrogen 17.1 and urea 36.6. Total protein 7.7 with an A:G ratio of 5.0:2.7. Blood chemistry: Chlorides 95, calcium 4.4, phosphorus 2.3, acid phosphates 3.6. Urinalysis normal. Mazzini and VDRL tests were negative. Chest X-ray negative for pulmonary, mediastinal or cardio-vascular pathology.

Course in Hospital: Because of the completely negative work-up in the hands of a very competent internist, because of the asthma-like picture, and not wanting to miss a possible tracheal tumor, I was asked to bronchoscope the patient. On July 16, 1953, preparatory to bronchoscopy the larynx was viewed with a mirror. Both vocal cords were in adduction with a glottic chink of 2 mm. or less. There was a minute

amount of motion of the arytenoids, and for the moment I felt that the patient might be holding the cords in adduction voluntarily. Certainly there was enough narrowing of the glottis to explain the stridulous, crowing respiration. Direct laryngoscopy was performed with the anterior commissure laryngoscope. The arytenoids and cords could not be abducted. A diagnosis of bilateral crico-arytenoid joint fixation (presumably due to arthritis) was made. Immediately following, a tracheotomy was performed to establish a more adequate airway. The patient was discharged on July 21, 1953, on a course of therapy including Cortisone in an effort to establish whether the crico-arytenoid joint fixation would respond to this form of treatment.

She was closely followed on an ambulatory basis. No increase in motion of the vocal cords developed after adequate therapeutic test, and so the patient was re-admitted to the hospital on Oct. 21, 1953, and a left arytenoidectomy was performed according to the method of DeBord[11] via laryngofissure. Her postoperative course was uneventful. She was discharged on Oct. 29, 1953, still wearing the tracheotomy tube. She was decannulated eight weeks later. The left vocal fold had retracted laterally so that the glottis at the posterior commissure was about 4 mm. wide.

Subsequent Course: For the next two years the patient, her husband, and her son (a medical student) were the source of considerable difficulty to me. The patient herself seemed to be in good health. Her voice was satisfactory, but apparently there was still occasional dyspnea on exertion, so that neither she nor her family was completely satisfied or happy, even though there were no episodes of stridor or dyspnea remotely resembling those which had occurred prior to arytenoidectomy. I was quizzed on one or two occasions by the medical-student son on the subject of "arthritis of the crico-arytenoid joint," and derived what little satisfaction I could from learning that *he* had never heard of it.

It was mutually agreed that perhaps it would be the wisest course to get an additional opinion on her status, and in September, 1955, this patient was seen in consultation by Dr. Chevalier L. Jackson, who was kind enough to discuss his findings with me. It was his opinion that the glottic airway was not quite wide enough for complete safety. It was his opinion, and I am thankful that I followed this advice, that perhaps the wisest thing would be to operate on the opposite, or right, arytenoid rather than attempt to do anything with the previously operated side. Accordingly, the patient was re-admitted to the hospital on Oct. 24, 1955, and following preliminary tracheotomy, the right arytenoid was resected via anterior thyrotomy (laryngofissure) as described by DeBord. She was discharged on Nov. 6, 1955, with the tracheotomy tube in place.

This time her postoperative course was somewhat complicated by the development of a polypoid mass, pedunculated, attached to the base of the epiglottis just above the anterior commissure, and covering the anterior half of the rima glottidis. On Dec. 12, 1955, this was removed via direct laryngoscopy. The second (right) arytenoidectomy bed had retracted laterally so that the glottis was now about 5 to 6 mm. at the posterior commissure, and while the voice was more "breathy" and not so strong, there was no further dyspnea or stridor. The patient was decannulated on Feb. 15, 1956, and since then has been getting along well.

Figs. 6 and 7 are histologic sections from the left and right arytenoids of Case 3 (above). They represent sections through the articular surfaces. Obviously, in doing an arytenoidectomy the crico-arytenoid joint *per se* is disarticulated, so a joint cavity cannot be shown as it was in Figs. 1 to 5; furthermore,

the average (and even better than average) pathology laboratory is not equipped or staffed for using the celloidin technique. Once a small specimen like the arytenoid is embedded in the usual paraffin block it is almost impossible to orient this block to the microtome knife so that serial sections are through the articular surface. It is for this reason that histologic sections are not available on every case; further-

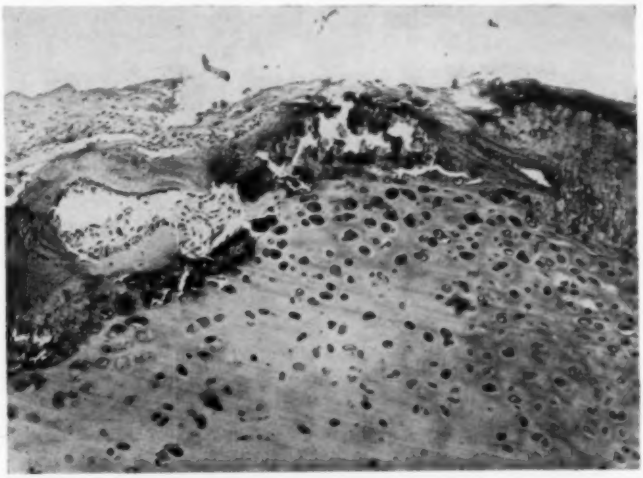

Fig. 6. Microphotograph (X125) of left arytenoid from Case 3. (This was the first arytenoid operated upon in this case). The articular surface is irregular in contour and shows tearing out of the tissue as a result of the microtome knife dragging through what was probably calcified surface. Many of the cartilage cells beneath this area are calcified. To the right of it lacunae and canaliculi are forming as bone develops. To the left may be seen a portion of the synovial joint space of the crico-arytenoid articulation. It is lined by endothelial cells. On the articular surface the cartilage cells show fragmentation and fibrillary changes in the few localized areas. These changes are compatible with those seen in degenerative arthritic changes. Diagnosis: Arytenoid cartilage showing changes compatible with arthritis.

more, unless the process of decalcification is carefully carried out, a paraffin block will not hold the specimen in its proper relationship as well as celloidin, making it difficult to obtain a good histologic section. For those which I have I am exceedingly grateful to Dr. Thomas Morrione, Head of the Department of Pathology at the Long Island College Hospital.

In all of the reference material cited in this thesis, the article by Pearson[35] contains the only histologic section of a crico-arytenoid joint. His patient died of an intercurrent broncho-pulmonary infection and the entire larynx was obtained at autopsy.

Case 4. J.H., a 28-year-old colored male was admitted to the Brooklyn Veterans Administration Hospital on Feb. 9, 1955, for evaluation and treatment because of "hoarseness and difficulty in breathing."

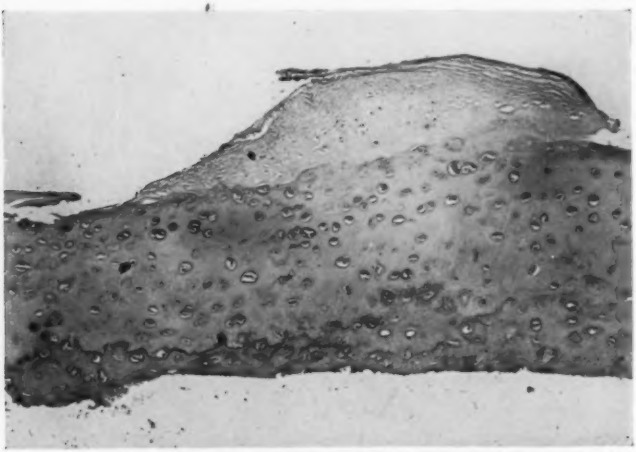

Fig. 7. Microphotograph (X125) of the right arytenoid from Case 3. (This was the second arytenoid operated upon in this case). This area shows an overgrowth of cartilage and fibrous tissue on one portion of the articular surface of the cartilage. Diagnosis: Arytenoid cartilage showing changes compatible with arthritis.

Present Illness: The patient stated that his present difficulty began in 1951, following an upper respiratory infection when he developed hoarseness and dyspnea which progressed in severity. He was in the Armed Forces at the time and had been sent to Alaska, but was returned to the United States and hospitalized at Walter Reed Army Hospital, where a diagnosis of "vocal cord paralysis" was made. He was subsequently discharged from the service on June 9, 1952, with a 10 per cent disability compensation for vocal cord paralysis. Since that time he has noted slowly progressive increasing dyspnea and hoarseness.

Past History was negative for diphtheria and typhoid fever. He had always been in good health except for his above-noted present illness. There was no history of neck trauma, no thyroid surgery and no other neurological symptoms. He denied ever having had venereal disease. There was no history of arthritis, rheumatism or joint pains.

Physical Examination revealed a well-developed, well-nourished colored

male who was slightly dyspneic at rest, this dyspnea increasing on exertion. Blood pressure was 125/75; the remainder of his general physical examination was entirely negative. Indirect laryngoscopy revealed a bilateral mid-line position of the vocal cords.

Laboratory and X-ray Data: Complete blood count was within normal limits; serological test for syphilis was negative; urinalysis was within normal limits. Blood uric acid was 3.7. The Rose test was negative. Chest X-ray was reported as negative for cardiovascular, pulmonary, pleural or mediastinal pathology.

Course in Hospital: On Feb. 24, 1955, in order to test for passive mobility of the cords, a preliminary tracheotomy was performed followed by direct laryngoscopy under general anesthesia. Pressure with the anterior commissure laryngoscope and with a closed laryngeal forceps revealed that both cords were fixed in the mid-line with bowing possible in the anterior two-thirds but no motion of the arytenoid cartilages. The glottic chink at the posterior commissure was approximately 2 mm. in width. On March 9, 1955, under general anesthesia a left arytenoidectomy was performed via laryngofissure approach according to the method of DeBord. By April 1, 1955, mirror laryngoscopy revealed that most of the edema had subsided from the larynx except for the anterior third of the left cord. The left cord at the posterior commissure was retracted approximately 4 mm. at this time. Incidentally, the patient was well enough on April 15, 1955, to have a circumcision performed. On April 26, 1955, he was sent on a 30-day leave of absence, following which indirect laryngoscopy revealed the airway to be quite adequate, with a posterior glottis of approximately 5 mm. The voice was breathy but satisfactory. The tracheotomy tube was removed, and the patient was discharged on May 27, 1955.

Subsequent History: The patient was re-admitted on Dec. 12, 1955, because of the development of a "tight scar" in the area of the previous tracheotomy. This was revised, and he was discharged on Jan. 5, 1956.

The patient was re-admitted on Nov. 13, 1957, because of increasing hoarseness and slight dyspnea of approximately ten days' duration. Indirect laryngoscopy revealed a considerable amount of diffuse polypoid hypertrophy of the left vocal fold so that the anterior two-thirds of the glottic chink was completely obstructed, and a relatively small aperture was present in the region of the posterior commissure. The posterior end of the left or previously operated vocal cord still appeared to be aprroximately 5 mm. away from the right arytenoid. Because of the dyspnea and poor cooperation on the part of the patient under local anesthesia, a preliminary tracheotomy was performed through the old scar, and via direct laryngoscopy under general anesthesia the left vocal cord was stripped. On Nov. 30, 1957, patient was decannulated. The voice and airway were satisfactory, and the patient was discharged on Dec. 9, 1957. He has had no difficulty since then.

Case 5. R.D., a 51-year-old white female was referred to me at the office on July 23, 1956, by Dr. Charles R. Weeth, Director, Department of Otolaryngology, Long Island College Hospital. In April, 1956, the patient developed dyspnea and hoarseness, or dysphonia, which increased over a period of several days, finally terminating in an episode of loss of consciousness with admission to a local hospital on an emergency basis. There she responded to rest, following initial oxygen therapy, and was discharged after 24 hours, somewhat improved, but still feeling that there was remaining shortness of breath. Incidentally, she gave a history of "asthma" for two years prior to this. The day after her discharge from this hospital, she developed acute respiratory distress with cyanosis and unconsciousness. She was taken to another hospital where an emergency tracheotomy was performed. She remained in the hospital for a period of

approximately ten days. The tracheotomy was performed by one of the thoracic surgeons on the staff. I do not know of any otolaryngologic consultation while she was in this hospital. Her physician had difficulty in decannulating her and she was discharged with the tracheotomy tube still in place. Following this, she was seen by the referring otolaryngologist who admitted the patient to the Long Island College Hospital on June 24, 1956, for direct laryngoscopy and removal of a polypoid pedunculated mass occupying the anterior half of a narrow glottic chink. Her laryngologist noted what he described as some abductor weakness, or incomplete abduction of the vocal cords at this time, but felt that her glottic chink was adequate and followed her on an ambulatory basis after discharging her on the following day. During the subsequent month, several attempts were made to decannulate the patient, and she apparently successfully tolerated complete closure of her tracheotomy tube for a number of days and was decannulated. On several occasions during this time, her laryngologist noted the lack of abduction on the part of both vocal cords, but with the partially obstructive polyp out of the way both he and the patient felt that she could get along, particularly in view of the fact that the tracheotomy tube had been corked for a number of days. About July 21, 1956, however, during a visit to his office, it became obvious that the glottic chink was not adequate, and because of his knowledge of my interest in cases of this nature, and suspecting a crico-arytenoid joint arthritis, he kindly referred the patient to me. I saw the patient in my office on that day, and indirect laryngoscopy revealed a glottic chink of 2 to 3 mm. at the posterior commissure with mild dyspnea, a fairly good voice and no cyanosis. I felt, however, that this was a dangerously small laryngeal airway and recommended that the patient have a tracheotomy.

She was admitted on my service on July 25, 1956, and a preliminary tracheotomy was performed with the insertion of a No. 6 tube. Following this, direct laryngoscopy under general anesthesia was done to test for mobility of the vocal cords and crico-arytenoid joints. The anterior two-thirds of the cord could be bowed; but both arytenoid cartilages were fixed, so that abduction was not possible. Arytenoidectomy was recommended, but the patient chose to delay this procedure temporarily, and she was discharged on July 27, 1956. At this time she was referred to an Internist for medical consultation and thorough work-up, and for control of an obviously active rheumatoid polyarthritis. She was seen by him on Aug. 21, 1956, for the first time, and thorough medical work-up was begun. She was seen by him again on Sept. 10, 1956, and Oct. 30, 1956, prior to re-admission for definitive surgery on the larynx. His history indicated that the patient had had arthritis for 12 to 13 years involving multiple joints. She had been on Cortisone and several other drugs, prescribed by preceding physicians taking care of her. In August of 1956, her chest X-ray was negative. His impression was that the primary problem was an active rheumatoid arthritis, and that gout had to be ruled out.

Laboratory findings were as follows: At the time of admission to the hospital for initial laryngoscopy and polypectomy and subsequently, her red blood cells ranged from 4.5 to 4.9 million; her hemoglobin between 11 and 13 gms. Her white count on several occasions ranged from 7,800 to 10,850 with 67 per cent polys in the former instance and 47 per cent polys in the latter instance. Serology was negative. Sedimentation rate on Aug. 23, 1956, was 57, at which time there were 9,150 white cells with 76 per cent polys. Her fasting blood sugar ranged between 100 and 124; urea nitrogen was 13.5, urea 28.6. Her urine was repeatedly normal. A C-reactive protein was reported slightly positive on Aug. 23, 1956. On the same date, uric acid was 3.2, total protein 7.4, serum albumen 4.7, serum globulin 2.7. X-ray studies on Aug. 23, 1956, revealed "generalized

arthritis involving the elbows, wrists, knees and small joints of the hands, characterized by hypertrophic periarthritic changes, cartilaginous necrosis, articular surface erosions and subarticular cystic degenerative changes."

The patient was treated medically principally with salicylates and ferrous gluconate, with codeine for severe pain.

She was readmitted to the Long Island College Hospital on Nov. 24, 1956, for steroid therapy prior to arytenoidectomy. On Dec. 3, 1956, a right arytenoidectomy was performed via thyrotomy; her post-operative course was uneventful, and she was discharged from the hospital on Dec. 12, 1956.

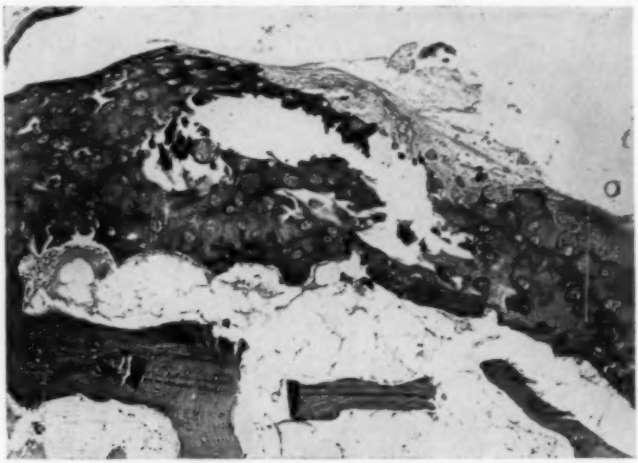

Fig. 8. Microphotograph (X125) from Case 5 of a section of arytenoid cartilage showing an articular surface covered with perichondrium that contains a very irregular contour in relation to the articular surface. Extensive fibrosis is present which dips down into the scalloped surface of the cartilage. Approximately 0.5 mm. below surface the cartilage is extenaively replaced by bone containing wide expanses of fatty marrow. The cartilage cells show some disturbance in their arrangement. The surface shows increase of fibrous covering. Diagnosis: Osteoarthritis of arytenoid joint surface.

She was then followed ambulatory in my office and on Jan. 29, 1957, the postoperative edema of the larynx had subsided, and the right vocal cord had retracted laterally so that the glottic chink at the posterior commissure was approximately 5 mm. across. The patient could tolerate complete closure of the tracheotomy tube for about one week prior to this date, so on that day, she was decannulated. Because of the fact that the tracheotomy tube had been in place from the latter part of July, 1956, to the latter part of January, 1957, a period of six months, a small tracheal fistula persisted, and the patient was readmitted on June 20, 1957, for plastic closure of the tracheal fistula. She was discharged on June 23, 1957.

This patient has been followed periodically regularly. She has an adequate airway and a serviceable voice which she uses to excess. She is being carried on small doses of Cortisone for her polyarthritis and is thoroughly rehabilitated.

The pathological report reads as follows: "Microscopic description: Section of the decalcified specimen shows that the cancellous bone is covered by cartilage which in turn is covered by layers of fibrous tissue. The cartilage cells show some disturbance in their arrangement. The surface shows an increase of fibrous covering. No fibrinoid changes are noted on the surface. Diagnosis: Osteoarthritis of the crico-arytenoid joint" (see Figs. 8, 9).

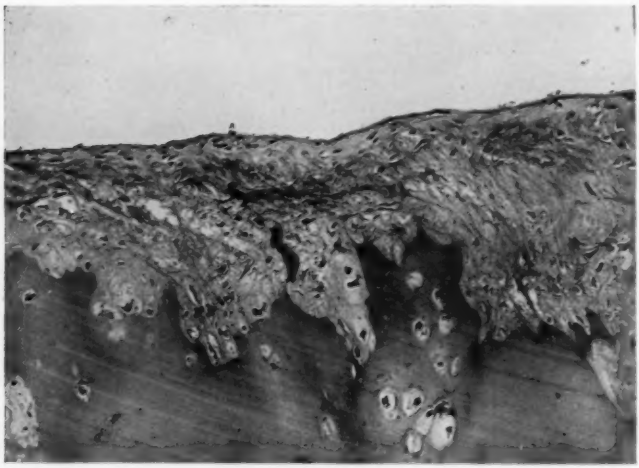

Fig. 9. Microphotograph (X90) of an area from the hip joint of a 52-year-old male. Diagnosis: osteo-arthritis. Note similarity of alteration in the articular cartilage compared with Figs. 6, 7, 8, including fibrous dyspiasia on surface aspect with scalloping of cartilage.

Case 6. W.E., a 25-year-old colored male was admitted to the Brooklyn Veterans Administeration Hospital on March 7, 1957, complaining of hoarseness of about one year's duration and increasing shortness of breath of about three or four weeks' duration. He had recently seen a local physician who had advised surgery.

Review of Systems was non-contributory.

Past History: He stated that he had had an occasional episode of "arthritis" in the past, which consisted principally of mild joint pains in the large joints such as the knees, wrists and elbows; however, there was no clinical evidence of arthritis at the time of his admission to the hospital in the nature of fever, redness, swelling or limitation of motion. There was no history to suggest diphtheria or typhoid fever. He denied ever having had any venereal disease. There was no history of trauma to the neck. There had been no thyroid or anterior neck surgery.

Physical Examination revealed a well-developed, well-nourished Negro male with moderate stridor, both on inspiration and expiration. Pulse was 64. Blood pressure 120/85. The remainder of the physical examination was entirely negative except for mirror laryngoscopy which revealed a midline position of both vocal cords with no abduction.

Laboratory and X-ray Data: A white count on admission was 11,100, hemoglobin was 13.2 gm. Urinalysis was normal. The cardio-lipin microflocculation test was positive in 1:8 titer. X-ray revealed no evidence of cardio-vascular, pulmonary, pleural or mediastinal pathology.

Course in Hospital: On March 22, 1957, the patient had a preliminary tracheotomy. On March 26, 1957, direct laryngoscopy was performed under general anesthesia administered via the tracheotomy tube. The anterior-commissure laryngoscope was introduced in order to test for mobility of the crico-arytenoid joints. Insertion of the tip of the scope in the anterior commissure resulted in a bowing of the anterior two-thirds of the cords with no lateral motion at the crico-arytenoid joints. These remained in the mid-line position. Following the preliminary test for passive mobility of the vocal cords a left arytenoidectomy was performed via a laryngofissure approach according to the method of DeBord. By April 17, 1957, the postoperative laryngeal edema had subsided sufficiently so that the tracheotomy tube was removed. The glottis at the posterior commissure at this time was approximately 5 mm. in width, which was adequate, and while the voice was somewhat "breathy" it was perfectly serviceable. The patient was discharged on May 2, 1957.

REVIEW OF LITERATURE AND COMMENTS.

As has been noted, at the time I saw my first case of ankylosis of the crico-arytenoid joint in 1952, a hasty check of the more recent American literature revealed only the case reported by Myerson[32] in 1943. It is interesting to note that the author seems to relate the development of a progressive bilateral crico-arytenoid ankylosis to an episode of peritonsillar abscess which preceded it by a period of several months. There was no similar antecedent history in any of my cases.

In December, 1955, Montgomery, Perone, and Schall[29] reported on four of six cases which they had seen. Their article directed my attention to American otolaryngological textbooks, in which they state, "arthritis of the crico-arytenoid joint is described."

For the most part, these textbook references are not extensive, yet they contain a great deal of information. For example, Boies[4] in the chapter on Hoarseness has a section titled "Fixation of Range of Movement of the Crico-arytenoid Joint," in which he states that this joint "may become partially or wholly fixed in its normal range of movement by inflam-

matory processes involving it from adjacent infection, trauma, or in an arthritic process such as may occur in any other joint. If the involvement is unilateral there may be relatively little interference with phonation and thus only slight, if any, hoarseness.

"The diagnosis of fixation is determined by testing the passive mobility of the arytenoid. This is done by palpating it with a blunt instrument through the direct laryngoscope. In cases of an obstructive bilateral fixation the joints may be mobilized through surgery."

Certainly there is a great deal of information in the foregoing, and for those willing to explore basic texts it is available.

Morrison,[30] on the other hand, has this to say under Affections of the Crico-arytenoid Joint: "Etiology: the arthritis is usually a destructive one which follows invasion of the joint by local infection and ulceration in tuberculosis and syphilis, contact ulcer, abscess of the arytenoid, perichondritis of the arytenoid or cricoid cartilages and trauma during direct laryngoscopy, bronchoscopy or forceps removal of tissue about the joint. It may occasionally be a rheumatic, non-suppurative process. Fixation follows suppuration in the joint because of scar formation and ankylosis."

Except for his reference to a rheumatic, non-suppurative process, neither my cases, nor those reported by Montgomery, et al.,[29], Copeman,[9] Pearson,[35] Baker and Bywaters[2] and Reaves[37] indicate that any of the other possible etiologic factors mentioned were involved; however, there is considerable material to support Morrison in the earlier, foreign and American literature[1,6,13,15,18,20,23,33,39] referring to the acute and chronic processes as being of etiologic significance.

Morrison[30] further states that "fixation of the cord is not amenable to any known form of treatment once the condition has become established." I presume that he refers here to treatment which will make the arytenoid move again. If this is so then I agree. Since he does not refer to bilateral midline fixation one cannot be at all critical; however, this reference might bear some clarification in future editions.

Continuing with American textbook references, we come to Chevalier Jackson[21] in which, in the chapter on Trauma of the Larynx the author, discussing ankylosis of the crico-arytenoid and crico-thyroid joints, says, "Traumatic ankylosis is more frequent than laryngeal literature indicates, but as compared to the incidence of ankylosis of these joints as a sequela of arthritic disease it is rare." This is a most interesting, and, at first blush, puzzling observation; however, it is nonetheless factual. This is because, while there is not a great deal in the recent literature on arthritis of the crico-joint, there is a fair amount in the literature of 30 to 65 years ago, while there is surprisingly little on traumatic ankylosis.

The same author, under Diagnosis, states that, "In crico-arytenoid ankylosis the absence of arytenoid movement is instantly recognizable in the mirror, but to make a diagnosis of ankylosis, recurrent paralysis must be excluded. This can be done readily by the Jackson 'passive mobility test'. In this test the closed laryngeal forceps are used through the laryngoscope to test passive mobility. The rigid fixity of the ankylosed arytenoid is strikingly different from its free passive mobility in cases of cordal paralysis."

One does not take issue with the foregoing. As a *confirmatory* test it puts the final seal of correctness on what should, by the time one gets to direct laryngoscopy in a suspected case, be a strong presumptive working diagnosis. The important point is that, as noted previously, one should rule out with a detailed history, those diseases which might conceivably cause crico-arytenoid fixation—such as diphtheria and typhoid fever (even though today these are rarely seen); rule out lues on history, examination and serology; rule out trauma, again with adequate history and physical examination; rule out thyroid or other low anterior neck surgery; rule out chest pathology which might involve the recurrent nerves (and while this is of great importance in unilateral cord immobility, particularly left-sided, it is almost inconceivable that bilateral recurrent paralysis without thyroid or other neck surgery, which should be obvious, could occur) again with history, physical examination and in addition

X-ray examination; rule out bilateral vagus nerve involvement (and so the recurrents) within the brain, by physical examination alone, and easily—for it is impossible to have a bilateral vagus nerve paralysis without involvement of at least one and probably not without all three of the following: Glossopharyngeal, spinal accessory and hypoglossal.

Having done all this in a case where there is bilateral mid-line position of the cords, it is my opinion that a strong presumptive diagnosis of *fixation* of the crico-arytenoid joint is justified, and on the basis of the experience with my six cases, this is true whether or not there is or has been, associated arthritic changes in any of the other joints of the body. It should be noted that in only two of these cases was there any history or findings to suggest polyarthritis.

This is the kind of history, physical examination and diagnostic reasoning which, I feel, make a good physician—the first requisite in making a good otolaryngologist, and one of the points which I wanted to make in this paper.

It goes without saying, I hasten to add, that one must still *confirm* the diagnosis, and this can be done simply by testing for passive mobility of the cords as described by the senior Jackson.

It would seem appropriate here to follow with the problem of further confirmation—by histologic section of the arytenoid removed at operation. We are all aware of the great importance of the work of hospital Tissue Committees. We are aware that the Combined Committee on Accreditation of Hospitals insists that these Tissue Committees oversee the material which comes out of operating rooms and passes through departments of pathology, so that they may check the agreement percentage between preoperative, postoperative and microscopic diagnoses. We have the anomalous situation, in connection with the pathological material presented in this paper, of my having had a rather difficult job in attempting to get the departments of pathology of the hospitals concerned, to provide me with histologic diagnoses and proper sections.

This is not a new problem for us as otolaryngologists, for we know that, in most instances, unless the Ear, Nose and

Throat Service at a particular institution has its own facilities for the preparation of histologic sections, much of the material with which we are vitally concerned, cannot properly be processed. This may not be too extensive a problem with regard to tissues sent to Pathology from operating rooms where, in most cases, the tissue is soft; but how often do we get unsatisfactory reports on hard tissues such as bone and cartilage (the latter in many cases having undergone ossification). Much more important, how often are we frustrated in attempting to get sections from autopsy material of temporal bones, sinus walls, and larynges? This, after all, is the very reason why the American Academy of Ophthalmology and Otolaryngology has seen fit to set up central laboratories for the processing and reading of histologic sections of temporal bones.

There is good and ample reason for this: The average hospital pathology department is not set up to process this kind of material. They dislike the tedium involved in decalcification, and the additional laboratory technical assistance required. Their material is embedded in paraffin because it is quicker and more simple. They are not equipped to embed tissues in celloidin. Even where the surgeon is interested enough to follow the tissue down to the pathology laboratory he finds it difficult to orient an ossicle, for example, or a cupola, or, as in this instance, an arytenoid cartilage, once it is embedded in paraffin, so that sections will arrive on slides in a manner best suited for useful histologic reading. Paraffin is practically opaque. Celloidin is relatively translucent—almost transparent; furthermore, with ossified material, paraffin will often not hold tissues in their proper relationship against the cut of the microtome knife. Celloidin usually will.

These are some of the problems which resulted in the fact that there is not a microphotograph for each of the cases reported herein. For those which I have I am exceedingly grateful; for those which are absent, my apologies.

Montgomery, Perone and Schall[29] in 1955, remark that there is nothing in the American literature on crico-arytenoid joint arthritis "in the past thirty years;" yet, included in their bibliography is the report of Myerson[32] in 1943. I mention

this, not as a criticism of their paper, for one can easily understand how an oversight of this kind might occur. Actually, the paper of Myerson[32] is the only one which has come to my attention in the American literature on laryngology in the 30 years from 1926 to 1955; however, not on the exact subject, but closely allied is an interesting report by Babbitt[1] in 1922, written in the formal narrative style of that day, in which he reports the . . . "Loss of Both Arytenoid Cartilages." Somewhere in the neighborhood of three weeks after major abdominal surgery and the postoperative use of a feeding tube, and, ". . . some eighteen days after my first examination, the patient coughed out what appeared to be the left arytenoid cartilage, and in two or three days the right one followed. X-ray, fluoroscope and laboratory examinations confirmed the diagnosis. This was further proved by conference with a professor of anatomy and one of our leading laryngologists. One specimen I present for examination; the other was sectioned at the laboratory. Two or three weeks following this, the patient commenced to recover voice, and after some six weeks was able to produce a fair tone without, however, much variation in pitch."

This case is cited for two reasons: first, because I suppose that if the arytenoid can undergo a morbid process which results in its sequestration there would seem little doubt that it may also be the seat of an arthritis resulting in ankylosis; second, because in Case 3 above, while the patient did not cough up her arytenoids, I removed both of them—and she has a serviceable voice. Actually this is because of the usurpative action of the ventricular folds, a dysphonia plicae ventricularis.

Another American reference is Harris[16] in 1919, in which he reports a case of ankylosis (spelled anchylosis at the time) of the crico-arytenoid joint on both sides requiring tracheotomy. He reports that anchylosis of the crico-arytenoid joint is a more common affection than is generally supposed, and that in its acute form it is undoubtedly often overlooked, while in its chronic form, when only one side of the larynx is involved, "without question" is often mistaken for recurrent nerve paralysis. He goes on, "Bilateral crico-arytenoid anchy-

losis is, however, a far rarer occurrence and one which, if the cords are fixed in the medial line, fraught with grave consequences to the life of the patient."

Newcomb[94] in 1893, in a journal no longer in existence, reports on "Rheumatism of the Crico-arytenoid Joint."

It was Casselberry,[7] however, as far back as 1893, in the Transactions of the American Laryngological Association, who wrote a most remarkable dissertation, scientific and somewhat philosophical, on the subject of "Arthritis Deformans of the Larynx." He concerns himself with the "propriety" of introducing this title (*i.e.*, Arthritis Deformans) into laryngeal nomenclature, and leaves its final disposition up to pathologists, as it may or may not relate to Arthritis Deformans in general, the disease also known as Rheumatoid Arthritis. He states that, "From careful studies of the morbid anatomy of arthritis deformans R. Volkmann (cited by Weber) recognizes the rapid proliferation of the articular cartilaginous elements, particularly on the free surface of the cartilage as the essential factor of the disease, together with subsequent absorption of the cartilages" (see Figs 6-9). He continues, "Garrod failed to discover any pathognomonic change in the blood, and Weber states that it is a fact that uric acid has not been found in the blood and that analysis of the urine has failed to show unusual increase in uric acid or urates." This has been true in the cases reported here as evidenced by the laboratory findings included in the case reports.

While the American literature has not contained much on this subject, the foreign literature has contained more; however, many of these references, as previously noted, deal with acute involvement of the crico-arytenoid joint, and most of them refer to unilateral involvement. They do not concern themselves so much with bilateral mid-line fixation of arytenoids and cords. Errecart,[13] for example, reports a case of gonococcal involvement of the crico-arytenoid joint. Haardt[15] deals with the clinical and pathological anatomy of perichondritis of the cricoid cartilage. Herzog[17] demonstrated a case of a 41-year-old male who had difficulty in breathing for ten years. Examination with a sound revealed bilateral crico-

arytenoid joint fixation. The glottis at the posterior commissure was 2 mm. wide.

Hiraoka[18] reports a case of acute rheumatism of this joint. Horbst[19] deals with the pathologic histology of arthritis of the crico-arytenoid joint. Hutter[20] speaks of the "localization" of acute arthritis in the larynx. Levinstein[23] refers to acute rheumatoid arthritis of the larynx. Litten[24] states that gouty depositions in the larynx are among the greatest rarities, and that in all of the gout literature there is only one reference by Virchow to a gouty tophus in the larynx.

Mygind[33] classifies affections of the crico-arytenoid joint in respect to etiology as follows: *1.* primary crico-arytenoid joint arthritis resulting from acute infections; *2.* primary crico-arytenoid joint arthritis resulting from chronic infections or toxic diseases; and *3.* various forms of rheumatic disease of the crico-arytenoid joint or in the immediate neighborhood of the crico-arytenoid joint.

Ritter[39] reports a case of acute rheumatoid arthritis with beginning symptoms referred to the larynx.

G. H. Mackenzie[25] in 1894 stated that, "It is only within a comparatively recent period that attention has been directed to the manifestations of rheumatism in and about the larynx. Probably the most conclusive proof of the accuracy of this assertion is that no mention of this condition is made by Morrell Mackenzie in his great work on diseases of the throat and nose, published in 1880 and 1884."

The above reference fascinated me, for in Morrell Mackenzie's[26] text on "Diseases of the Pharynx, Larynx and Trachea," published in 1880, the author has the following to say, and considering the fact that it was written almost 80 years ago one cannot help but marvel at the clinical acumen of our forebears. I quote from it almost *in toto* because there has been very little said in the literature after this which he does not say here.

The section of this book titled "Anchylosis of the Arytenoid Articulations" contains the following footnote:

"This article would have been more appropriately inserted

after perichondritis, but having been accidentally omitted in its proper place I have thought it better to insert it here, affections of the joint being so likely to be mistaken for muscular paralysis."

The section continues:

"Notwithstanding the exposed position of the crico-arytenoid articulation fixature of the joint must be rare; for immobility of the vocal cords is not a very common condition, and when it does exist it is often due either to nervo-muscular affections or to general tumefaction of the soft parts which mechanically prevent the movements of the cords. That it does occasionally occur there is little doubt. This subject has not been hitherto treated with any detail, although the condition has been incidentally referred to by Turck,[49] Sidlo,[45] Ziemssen,[52] Mandl,[27] Schroetter,[41] Koch,[22] Burow, Jun[5] and Semon.[43] Anchylosis of the crico-arytenoid joint may arise from perichondritis or chondritis, either of which may occur primarily or result from extension of disease from the superadjacent soft parts. It is probable also that it may be due to primary synovitis either rheumatic, gouty or simply catarrhal, and in some cases it most likely arises from mere disuse brought about either through the muscles having been previously paralyzed or through changes in the contiguous parts preventing the movement of the joint for a long period and thus giving rise to permanent anchylosis; or the fixature may be due to traumatic injuries such as wounds, contusions or dislocations. Perichondritis, generally due to typhoid fever or syphilis, is undoubtedly the most common cause of the affection, for according to Dr. Semon, out of ten cases on record, in five instances the disease was due to inflammation of the perichondrium covering the cricoid or arytenoid cartilages. The symptoms vary according as the disease is unilateral or bilateral, according to the degree of mobility of the joint, and according to the position in which the arytenoid is fixed on the cricoid cartilage. Thus, if the arytenoid be fixed on the outer part of the cartilage we have the vocal cord permanently drawn aside and permanent dysphonia, whilst if the arytenoid cartilage is fixed near the center of the cartilage, the vocal cord

is permanently fixed near the medianline, and there is persistent dyspnea.

"The diagnosis of the condition is attended with some difficulty, and paralysis of either the adductors or the abductors may simulate anchylosis. The affection may, however, be inferred to exist when immobility of one or possibly of both vocal cords is accompanied by some irregularity in the form of the cartilages or the upper part of the cricoid cartilage. It should also be especially looked for in the case of patients who are convalescent from typhoid fever and have some alteration in voice or difficulty in breathing. I am not aware that any treatment would be likely to give very satisfactory results, but if the arytenoid cartilages are fixed in a central position forcible dilatation should be affected according to the mechanical methods laid down in the last article, after tracheotomy has been performed; and this treatment should, if possible, be employed prophylactically in cases of perichondritis after typhoid fever and syphilis, in which considerable destruction of the joint has taken place, and subsequent anchylosis is to be feared."

One need only remark that at that time typhoid fever and untreated syphilis were much more common than now; and, secondly, that the operation for arytenoidectomy had not been described at that time.

Copeman[9] in a much more recent article remarks that, "There appears to be no reason why rheumatoid disease may not affect the crico-arytenoid joints in similar fashion to other larger diarthrodial joints of the body. If this is true, we should expect to observe cases which exhibit phases of recurrent acute or subacute inflammatory reaction either confined to these joints or, more probably, as part of a generalized arthritic process. In the later chronic stages of the disease, and usually as the sequel to recurring episodes of this nature, more permanent impairment of movement or even ankylosis of the crico-arytenoid joints might occur."

This is very well expressed, and even though in only two of the cases described in my paper was there any evidence of polyarthritis, I feel that the same sort of intermittently pro-

gressive process may well occur, to result in increasing impairment of mobility of the crico-arytenoid joint. How, otherwise, can one explain the relatively long and intermittent histories in every case of mine, prior to the final episode which resulted in truly disabling dyspnea? In many instances I feel that earlier acute or subacute exacerbations of joint pathology were probably responsible for the diagnosis of "asthma." In these stages anti-asthmatic therapy was to no avail. Why would it be? In these stages, while there is still *some* motion at the joint on mirror laryngoscopy, even the best of us are likely to mistake the incomplete abduction for the kind one so often sees in a patient who is anxious and not fully cooperative; furthermore, is this not the usual natural history of generalized rheumatoid arthritis—with its spontaneous exacerbations and remissions, and an intermittently progressive deformity and increasing fixation of whatever joint may be involved? While one might expect to see some remission on steroid therapy in an acute exacerbation, is there any reason to suppose that this kind of therapy will result in increased motion of a joint which has already become completely ankylosed? Cortisone had no effect in any of the cases on which I have reported.

Copeman[9] continues: "The differential diagnosis in these and similar cases would appear to lie between rheumatoid arthritis involving the crico-arytenoid joints and paralysis of the recurrent laryngeal nerves in rheumatoid subjects due to some unknown local or general cause of which no evidence can be found. The distinction, which can finally be made only by a *laryngologist of experience* (italics are mine), remains difficult and perhaps uncertain."

I find it hard to agree that bilateral abductor paralysis cannot be easily excluded in this differential diagnosis. It cannot occur centrally without other neurological symptoms and signs; it cannot occur peripherally except following thyroid surgery, or where there is intrathoracic (left-sided) disease *and* low cervical (right-sided) disease of a nature which would involve the recurrent nerves. Nor can I disagree when he states that the final distinction can be made only by a laryngologist of experience.

It should be noted, that while the article by Snell[46] does not deal with pathology involving these joints, it is included here because it is an extremely good treatise on the function of the crico-arytenoid joints in the movements of the vocal cords. Since it appears in a journal which is not likely to come before the otolaryngologist I take the liberty of calling attention to it here.

It remains now only to call attention to a recent article by Shambrom and Feher[44] in which these authors report a case of Tietze's syndrome with involvement of the crico-arytenoid joint. They are not aware of this association having been reported before. Tietze's syndrome is a benign, self-limiting disease characterized by tender, non-suppurative swelling over the upper costal cartilages. Usually one of the second costal cartilages is involved; multiple involvement of costal cartilages is uncommon. Pain usually persists for a few days to a few weeks while swelling may continue for months or even years. According to Motulsky and Rohn[31] its cause is not known; non-tuberculous respiratory disease or rheumatic conditions frequently precede or accompany the syndrome. On an anatomic basis, they suggest that small tears or rheumatoid involvement of the inconstant inter-articular sterno-costal ligament may be a factor in the etiology.

Shambrom and Feher[44] believe that involvement of the crico-arytenoid joint in their patient with Tietze's syndrome suggests that the condition can be more disseminated in the body than was previously thought. They state that, as more cases are described it is probable that more areas of the body (*i.e.*, more joints) will be found to be involved.

Attention is called to this syndrome for several reasons: first, if as noted, it may involve the crico-arytenoid joint, then as laryngologists we should be familiar with it; second, the clinical syndrome described is not remarkably different from rheumatoid arthritis elsewhere in the body; third, while perhaps not a diarthrodial articulation, the sternocostal junction is an articulation.

I wonder, therefore, whether this syndrome is not really another manifestation of rheumatoid arthritis, just as may

occur in the crico-arytenoid joints alone, and whether the case reported by Shambrom and Feher[14] with sterno-costal and crico-arytenoid joint involvement, is not a frequently overlooked variation of the multi-faceted, generalized or localized morbid process called rheumatoid arthritis.

SUMMARY AND CONCLUSIONS.

The anatomy, gross and histologic, of the crico-arytenoid articulation has been reviewed.

Evidence that this is, and should be thought of as, a true diarthrodial joint is presented.

Six cases are reported in which there was bilateral ankylosis of the crico-arytenoid joint with fixation of the vocal cords in the mid-line and resultant dyspnea requiring tracheotomy.

This ankylosis was the result of rheumatoid arthritis.

In four of these cases there was no evidence that any joints other than the two crico-arytenoid joints were involved.

Important points in the differential diagnosis have been presented.

The condition is amenable to surgical correction. This is to say that the airway may be made adequate, and a serviceable voice may be retained. Medical management is not adequate.

The treatment consists of the required preliminary tracheotomy, followed by any one of the various operations described for arytenoidectomy or arytenoidopexy.

The literature appertaining hereto has been reviewed and comments made thereon.

REFERENCES.

1. Babbitt, J. A.: A Mixed Infection of the Larynx, Followed by the Loss of Both Arytenoid Cartilages. *Trans. Coll. of Phys. of Philadelphia*, 44:383-384, 1922.

2. Baker, O. A., and Bywaters, E. G. L.: Laryngeal Stridor in Rheumatoid Arthritis Due to Crico-Arytenoid Joint Involvement. *Brit. Med. Jour.*, 2:1400, June 15, 1957.

3. Ballenger, H. C., and Ballenger, J. J.: "Diseases of the Nose, Throat and Ear," 10th Ed., p. 376. Lea and Febiger, Philadelphia, 1957.

4. Boies, L. R.: "Fundamentals of Otolaryngology," 2nd Ed. W. B. Saunders Co., Philadelphia, 1955.

5. Burow, J.: Cited by Mackenzie, M.[25]

6. Calamido, U.: Arthrites Crico-aritenoidiens Rhumatismales. *Arch. Int. de Laryngologie, D'Otologie et de Rhinologie*, 33:730-733, 1912.

7. Casselberry, W. E.: Arthritis Deformans of the Larynx. *Trans. Amer. Laryngol. Assoc.*, (15th annual meeting), p. 18-23, 1893.

8. Compaired: "Sobre La Artritis Crico-aritenoidea, aguda y subaguda, de Naturaleza reumatica," p. 163-166. El Siglo Medico, Madrid, 12 de Marzo de 1893.

9. Copeman, W. S. C.: Rheumatoid Arthritis of the Crico-arytenoid Joints. *Brit. Med. Jour.*, 2:1398-1399, June 15, 1957.

10. Cornelli, G. Artrite Cricoaritenoidea e Parisi Degli Abduttori. *Oto-Rino-Laring., Ital.*, 16:221-222, 1948.

11. DeBord, B. A.: Paralytic Stenosis of the Larynx; A Modification of the Intralaryngeal Approach to Arytenoidectomy. The Laryngoscope, 63:757-777, 1953.

12. DeVido, G., and Ancetti, A.: L'artrite Cricoaritenoidea, Quale Causa di Paralisi Laringea in Adduzione. *Minerva Otorhinolaryngol.*, 2:479-484, 1952.

13. Errecart, P. L.: Artritis Gonoccica de la Articulacion Crico-aritenoidea. *Rev. Asoc. Med. Argent.*, 49:1031-1033, July, 1935.

14. Goss, C. M.: "Gray's Anatomy of the Human Body," 26th Ed., p. 1204-1209. Lea and Febiger, Philadelphia.

15. Haardt, W.: Zur Klinik und Pathologischen Anatomie der Perichondritis des Ringknorpels. *Zeit. f. Hals-Nasen- und Ohrenkeilkunde*, 16:184-201, 1926.

16. Harris, T. J.: Anchylosis of the Cricoarytenoid Articulation with the Report of a Case Presenting Involvement of Both Joints and Requiring Tracheotomy. The Laryngoscope, 29:139-143, March, 1919.

17. Herzog: Case Demonstration No. 4, at Wissenschaftliche Aerztegesellschaft in Innsbruck. *Wiener Klin Woch.*, 31:1048, March, 1918.

18. Hiraoka, R.: Ein Fall von akutem Rheumatismus der Articulatio Crico-arytenoidea. *Oto-Rhino-Laryngol.*, 12:578, July, 1939.

19. Horbst, L.: Zur Pathologischen Histologie des Kriko-arytaenoid-gelenkes. *Monatssch. f. Ohrenk.*, 70:48-57, Jan., 1936.

20. Hutter, F.: Ueber die Lokalisation des akuten Gelenks-rheumatismus im Kehlkopf. *Wiener Klin. Woch.*, 35:247-248, 1922.

21. Jackson, C.: "Trauma of the Larynx." Otolaryngology (loose-leaf), Vol. IV, Chapt. 33, pp. 1-7. W. F. Prior Co., Hagerstown, Md., 1957.

22. Koch: Cited by Mackenzie, M.[25]

23. Levinstein, O.: Uber den akuten Gelenksrheumatismus des Kehlkopfs. *Zeit. f. Laryngol., Rhinol.*, 8:365-374, 1919.

24. Litten, M.: Pathologisch-anatomische Beobachtungen: Ein Fall von Schwerer Gicht mit Amyloiddegeneration. *Virchow's Arch.*, 66:129-139, 1876.

25. Mackenzie, G. H.: Rheumatism of the Larynx. *Edinburgh Med. Jour.*, 40:507-509, 1894.

26. Mackenzie, M.: "Diseases of the Pharynx, Larynx and Trachea," p. 347. Wm. Wood & Co., New York, 1880.

27. Mandl: Cited by Mackenzie, M.[26]

28. Maximow and Bloom: "Textbook of Histology," 6th Ed., p. 140. W. B. Saunders Co., Philadelphia, 1952.

29. Montgomery, W. W.; Perone, P. M., and Schall, L. A.: Arthritis of the Crico-arytenoid Joint. *Ann. Otol., Rhinol. and Laryngol.*, 64:1025-1033, 1955.

30. Morrison, W. W.: "Diseases of the Ear, Nose and Throat," p. 582. Appleton-Century-Crofts, Inc., New York, 1955.

31. Matulsky, A. G., and Rohn, R. J.: Tietze's Syndrome. *Jour. A.M.A.*, 152:504-506, 1953.

32. Myerson, M. C.: Fixation of Cricoarytenoid Joints Due to Streptococcic Infection: Improvement with the King Operation. *Arch. Otolaryngol.*, 38:169-170, June, 1943.

33. Mygind, S. H.: Ueber Arthritis Crico-arytenoidea Rheumatica Acuta und mit Derselben Verwandte Larynxleiden. *Arch. f. Laryngol. und Rhinol.*, 28:45-59, 1913.

34. Newcomb, J. E.: Rheumatism of the Crico-arytenoid Joint. *Ann. Ophthal and Otol.*, 2:347-349, 1893.

35. Pearson, J. E. G.: Rheumatoid Arthritis of the Larynx. *Brit. Med. Jour.*, 2:1047, May, 1957.

36. Pressman, J. J., and Kelemen, G.: Physiology of the Larynx. *Physiol. Rev.*, 35:506-554, July, 1955.

37. Reaves, R. G.: Bilateral Crico-arytenoid Ankylosis (Surgical Treatment. *Arch. Otolaryngol.*, 65:603-605, June, 1957.

38. Rehank, P.: Uber Arthritis Arytenoidea. *Monatssch. f. Ohrenk.*, 69:152-154, Feb., 1935.

39. Ritter, H. U.: Ein Fall von Akutem Gelenks-rheumatismus mit Beginn der Erkraukung in den Kehlkopfgelenken und ein Fall von Kehldeckelzyste. *Munchener Mediz. Woch.*, 69:1051, 1922.

40. Saunders, W. H.: Crico-arytenoid Ankylosis or Laryngeal Paralysis? *Arch. Otolaryngol.*, 63:260-261, 1956.

41. Schroetter: Cited by Mackenzie, M.[26]

42. Scott-Brown, W. G.: "Diseases of the Ear, Nose and Throat," Vol. 1, pp. 615-616. Butterworth Co. Ltd., London, 1952.

43. Semon, F.: Cited by Mackenzie, M.[26]

44. Shambrom, E., and Feher, A.: Tietze's Syndrome; Report of a Case with Involvement of the Crico-arytenoid Joint. *Arch. Int. Med.*, 96:697-699, 1955.

45. Sidlo: Cited by Mackenzie, M.[26]

46. Snell, A. R. D.: On the Function of the Crico-arytenoid Joints in the Movements of the Vocal Cords. *Proc. Koninklizke Acad. Van Wetenschappen*, 50:1370-1381, 1947.

47. Thomson, St. C., and Negus, V. E.: "Diseases of the Nose and Throat," 5th Ed., p. 695. Appleton-Century-Crofts, Inc., New York, 1947.

48. Todd, T. W.: "Cunningham's Texbook of Anatomy," 7th Ed., p. 655. Oxford University Press, New York, 1937.

49. Turck: Cited by Mackenzie, M.[26]

50. WOODMAN, DEG.: A Modification of the Extralaryngeal Approach to Arytenoidectomy for Bilateral Abductor Paralysis. *Arch. Otolaryngol.*, 43:63-65, 1946.

51. WOODMAN, DEG.: Open Approach to Arytenoidectomy for Bilateral Abductor Paralysis with Report of 23 Cases. *Ann. Otol., Rhinol. and Laryngol.*, 57:695-704, 1948.

52. ZIEMSSEN: Cited by Mackenzie, M.[28]

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Interested physicians should write direct to the Department of Otolaryngology, 1853 West Polk Street, Chicago 12, Ill.

PURE-TONE THRESHOLD PATTERNS OBSERVED IN FUNCTIONAL HEARING LOSS.

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The purpose of this article is to clarify and expand information relating to the pure-tone threshold configurations and degrees of impairment observed in patients who present hearing loss of a functional nature. The Audiology and Speech Correction Clinic of the San Francisco Veterans Administration Hospital serves as a Veterans Administration referral center for patients with difficult diagnostic problems, many involving functional hearing loss. Continued experience with these patients and with routine referrals has provided us with clinical data that illustrate the dynamics of functional pure-tone threshold patterns.

Some definitions and a review of pertinent literature are essential to an understanding of the material that will follow. A functional hearing loss may be defined as a decrease in auditory threshold without organic basis. Pure functional hearing loss is relatively uncommon. When a patient presents an organic hearing loss elaborated in magnitude (either consciously or unconsciously) he is said to have a functional overlay to his organically based loss. Functional overlay is probably the most prevalent type of functional loss encountered in clinical practice. Functional symptoms occur frequently in organ systems that have had genuine pathology, hence it is not surprising to find that the preponderance of functional losses have an underlying organic component, or a history of some complaint relative to the ears.

There have been a limited number of published reports

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describing functional hearing loss patterns. Semenov[1] reported that "Practically all cases of psychic deafness have flat audiometric profiles . . ." Fournier[2] reported a flat pure-tone threshold configuration as typifying "malingering." Doerfler,[3] however, observed that 80 per cent of the functional cases in his experience presented a "saucer-shaped" audiometric pattern rising at both ends of the audiogram and lying between 50 and 90 db. He attributed this phenomenon to adherence to an equal-loudness standard. Johnson, Work and McCoy[4] pointed out that the functional saucer audiogram is not so prevalent as Doerfler had indicated, and that it appears primarily in patients who have no significant organic hearing loss, or in cases where there is a conductive loss underlying the functional component. Carhart[5] recently observed that ". . . a trough-shaped audiogram which tends to follow an equal-loudness curve, *i.e.*, an equal phon contour, immediately suggest (sic) the possibility of a non-organic hearing loss." It should be noted that in the frequency range usually measured clinically the phon contours do not strongly resemble saucers. The essential point here is that the patterns described above as characteristic of functional hearing loss were attributed to adherence to an equal-loudness standard.

ORGANICALLY-BASED SAUCERGRAMS.

It is important to maintain proper perspective and recognize that the audiometric patterns just described appear clinically on a purely organic basis. Several examples should illustrate this point. The first example (see Fig. 1, Case B.L.) is that of a 42-year-old white male who experienced a rapid unexplained perceptive hearing loss in his left ear. The right ear remained normal. Stenger and galvanic skin response (GSR) procedures corroborated the organic nature of the loss. The air-conduction audiogram of his left ear is similar to the functional pattern described by Doerfler. The second example (see Fig. 2, Case G.B.) is that of a 39-year-old white male with bilateral labyrinthine hydrops. The threshold configuration of his right ear shows a perceptive loss of the type described by Johnson, Work and McCoy. The third example (see Fig. 3, Case D.O.) is that of a 38-year-old white male with a mixed perceptive deafness secondary

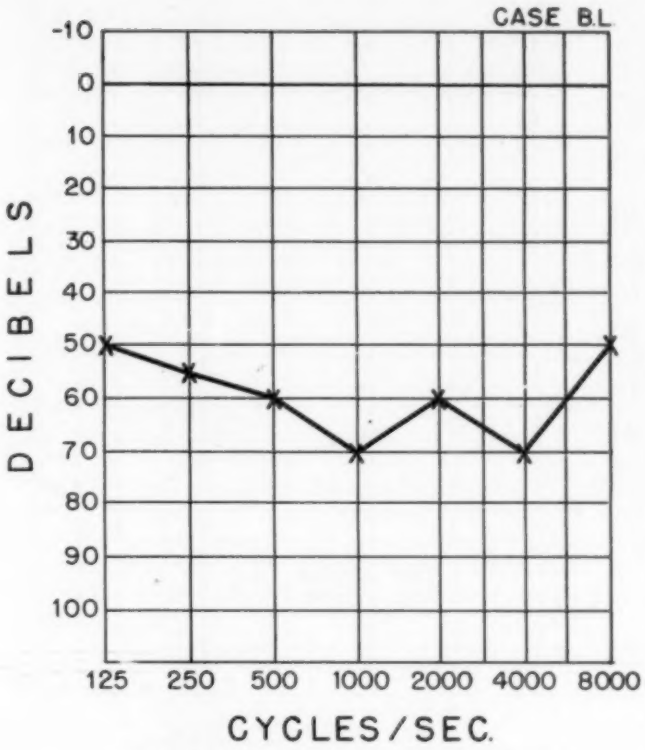

Fig. 1. Case B.L., Unexplained perceptive loss.

to advanced otosclerosis. The saucer-shaped audiogram of his left ear and the first two examples suggest that the shape of the audiometric curve is probably a poor single index of the presence or absence of a functional hearing loss. GSR audiometry at 1,000 cps confirmed the organic nature of the loss in each of these cases. An analysis of the functional cases in our clinic files revealed that only seven out of 190 ears (3.7 per cent) had pure-tone threshold patterns that were saucer-shaped. Saucergrams represent only one of the many audiometric patterns that may appear in patients who present a functional type of hearing loss.

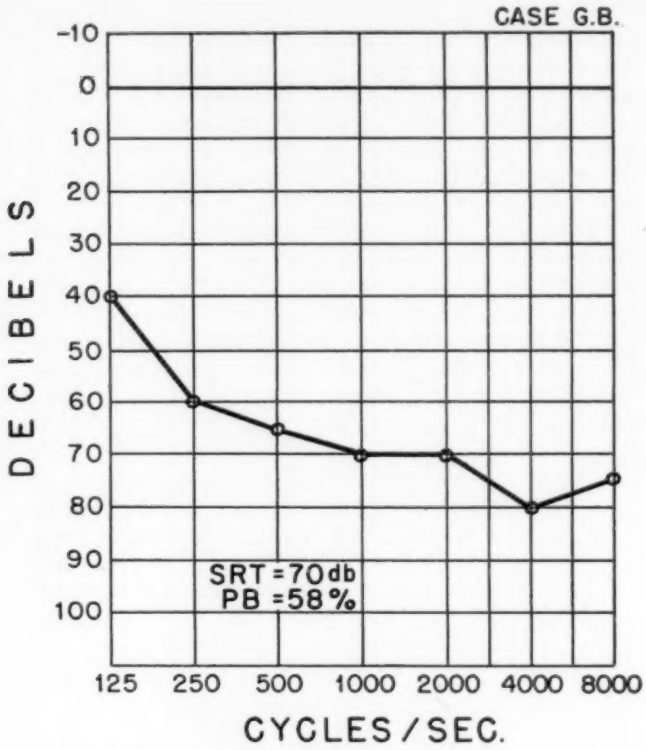

Fig. 2. Case G.B., Labyrinthine hydrops.

FUNCTIONAL PURE-TONE CONFIGURATIONS IN THE PRESENCE OF CONDUCTIVE LOSS.

In the assessment of routine cases we conduct GSR audiometry at 1,000 cps. In the assessment of functional cases, however, GSR audiometry is conducted at 500, 1,000 and 2,000 cps, and in some cases at 4,000 cps. GRS audiometry for all cases reported in this paper employed strict conditioning and response criteria with independent analyses of GSR graphic records by two audiologists. GSR testing has produced data illustrating some of the basic relationships between

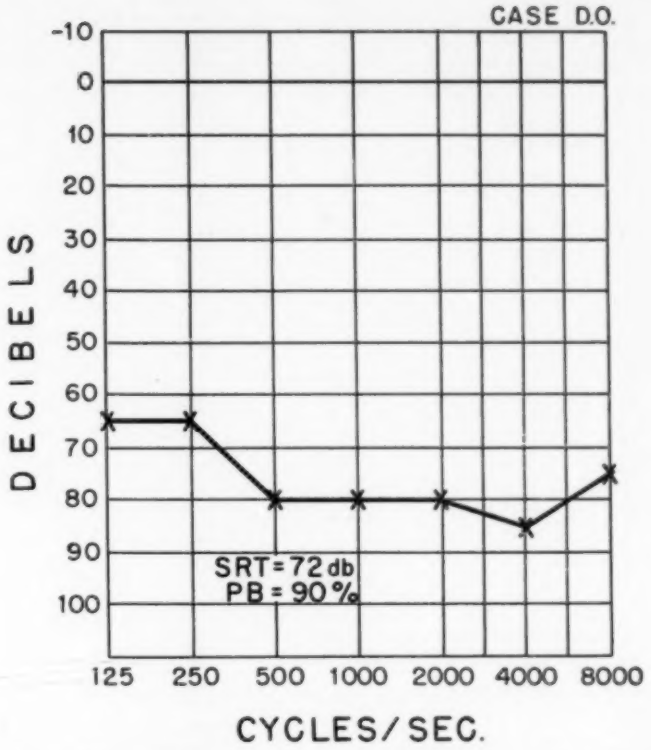

Fig. 3. Case D.O., Mixed perceptive deafness secondary to advanced otosclerosis.

type of organic loss, extent of functional component, and shape of the audiometric curve. An examination of some of these cases should help to illustrate some of the variables that operate in functional hearing loss.

The two cases that follow indicate that pure-tone audiometric patterns other than saucergrams occur in the presence of flat conductive hearing loss. This is contrary to the hypothesis advanced by Johnson, Work and McCoy.

The first of these cases is that of a 42-year-old white male laborer with a childhood history of painful otitis media and a reported decrease in

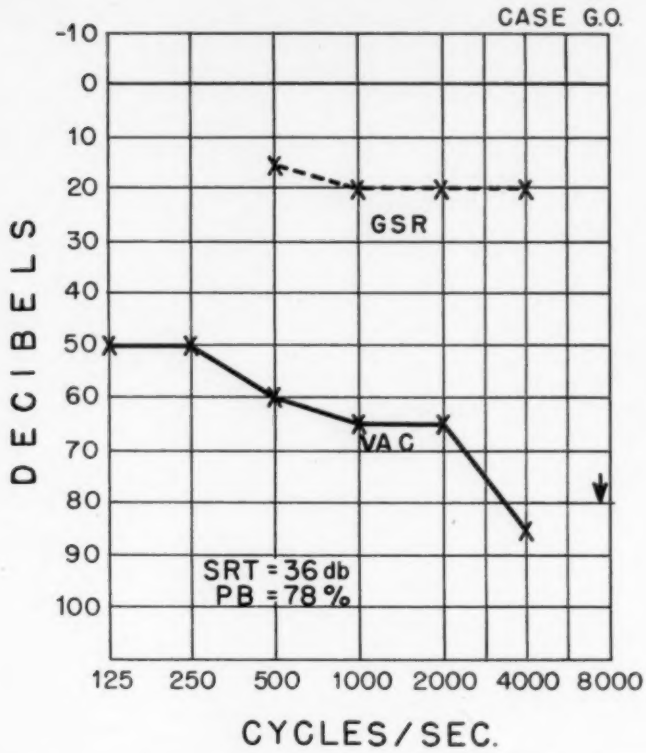

Fig. 4. Case G.O., Drop at 4,000 cps in voluntary air-conduction curve (VAC) with flat organic base (GSR).

hearing on exposure to mortar fire during service. Physical examination revealed slightly scarred drums with calcific plaques. GSR audiometry (see Fig. 4, Case G.O.) shows a bilateral minimal decrease in hearing acuity. Considering the medical history it is likely that in the range measured by GSR the loss is a flat conductive loss, yet his voluntary air-conduction curve at 4,000 cps falls rather than rises. The speech reception threshold (SRT)* shown in Fig. 4 for this case is considerably lower than the average loss in the speech frequencies. We have found this to be a typical intertest relationship in patients with functional hearing loss. We have, then, an example of a gradually falling curve in the presence of an essentially flat organic base.

*SRTs reported in this article were assessed with the CID W-1 spondaic word lists and intelligibility was measured with the CID W-22 word lists. SRTs and PB scores were obtained at the time of the first pure-tone audiogram.

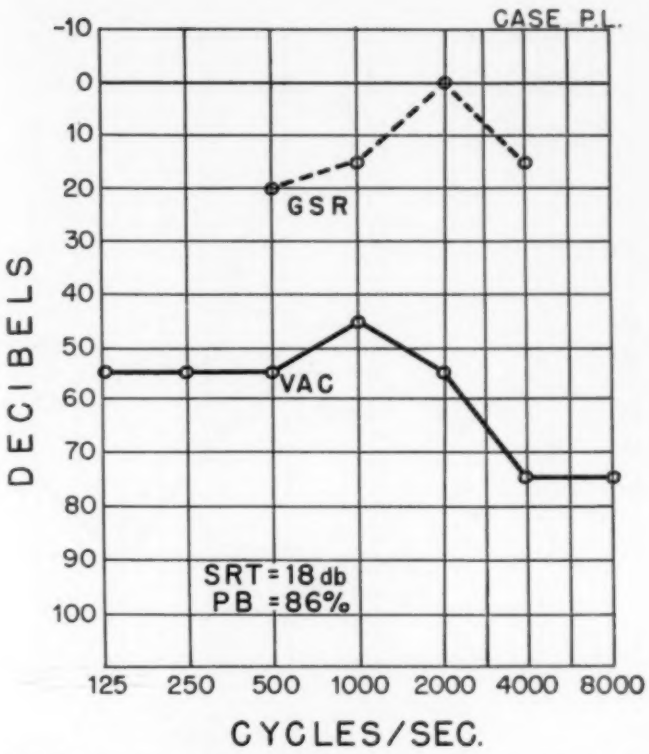

Fig. 5. Case P.L., Sloping voluntary air-conduction curve (VAC) with conductive organic base (GSR).

The next case (see Fig. 5, Case P.L.) illustrates a similar situation in a 22-year-old white male farm worker with a bilateral functional overlay. Medical examination revealed both tympanic membranes were scarred, and each had a large central perforation covered with a thin pseudomembrane retracted against the promontory. The GSR thresholds for his right ear demonstrate an essentially flat loss (excluding 2,000 cps) which is probably conductive in nature. The voluntary pure-tone audiogram is relatively flat until it drops 20 db at 4,000 cps without any apparent underlying organic basis for such a drop.

THE LIMITING EFFECTS OF RECRUITMENT ON THE EXTENT OF FUNCTIONAL OVERLAY.

Johnson and his colleagues described the effects of recruit-

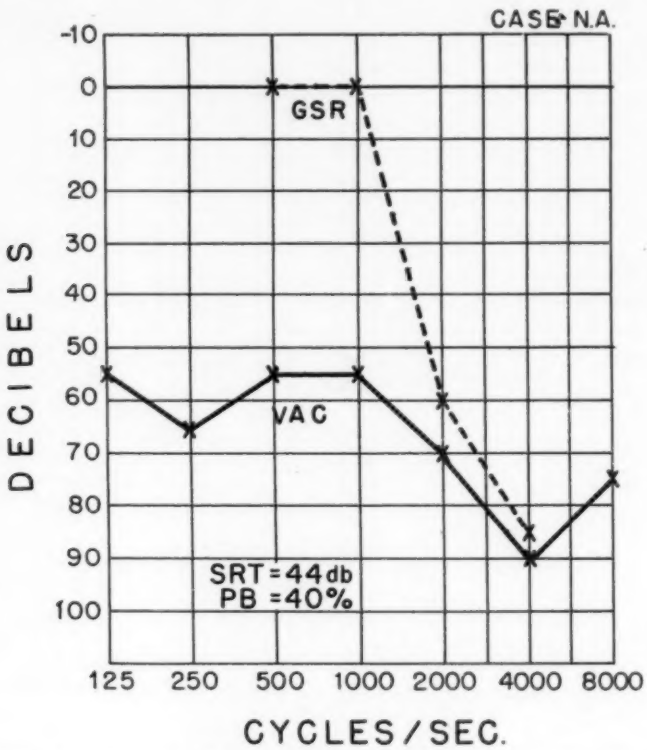

Fig. 6-a. Case N.A., Restriction of overlay at 2,000 and 4,000 cps where voluntary air-conduction thresholds (VAC) approximate GSR thresholds.

ment in limiting the extent of overlay at frequencies where there is a significant perceptive component to the loss. In their discussion of this phenomenon they expressed the opinion that the shape of the saucer audiogram is obscured in the presence of high-frequency perceptive loss. GSR audiometry offers an opportunity to provide objective evidence that functional overlay is restricted in the presence of perceptive hearing loss marked by recruitment. The cases presented in this section will offer supporting evidence for this hypothesis.

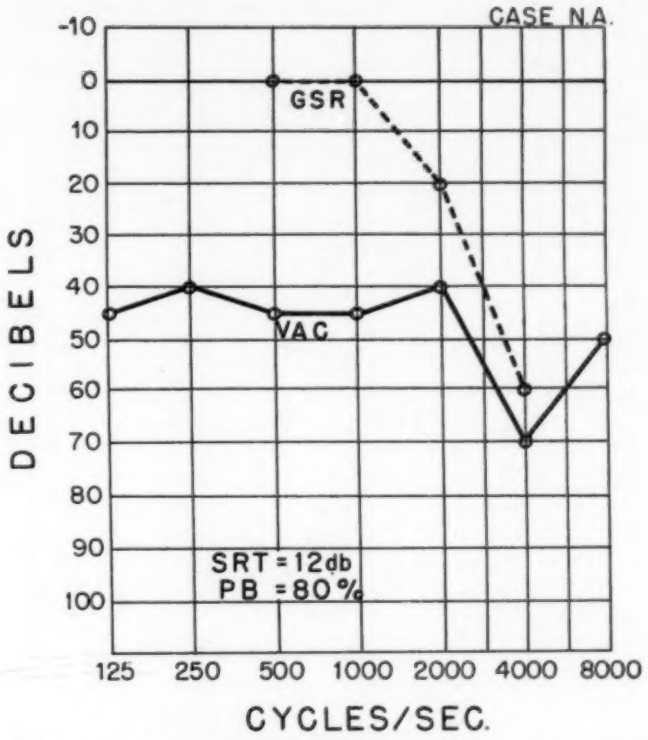

Fig. 6-b. Case N.A., Possible relationship between degree of recruitment and extent of overlay illustrated by differential in overlay at 2,000 and 4,000 cps. VAC = voluntary air-conduction.

The first example is that of a 40-year-old white male who gives a history of being thrown into the air by a large demolition explosion, after which he reported bilateral tinnitus, severe hearing loss in his left ear, and relatively normal hearing in his right ear. The psychiatric diagnosis in his case was schizophrenic reaction, paranoid type, in almost complete remission. His otological diagnosis was high-tone perceptive hearing loss, secondary to blast injury, with functional overlay. In the discussion of audiometry for this and succeeding cases, a basic assumption is that, in the presence of perceptive hearing loss, recruitment is a plausible explanation for the narrowing of functional overlay, particularly at the higher frequencies. An examination of the left ear test results (see Fig. 6-a, Case N.A.) of this patient shows that in the lower frequencies there is a 55 db functional overlay, yet at 2,000 cps there is only a 10 db overlay, and at 4,000 cps the overlay is only 5 db. At both of these frequencies the loss appears to be perceptive in nature, and the phenome-

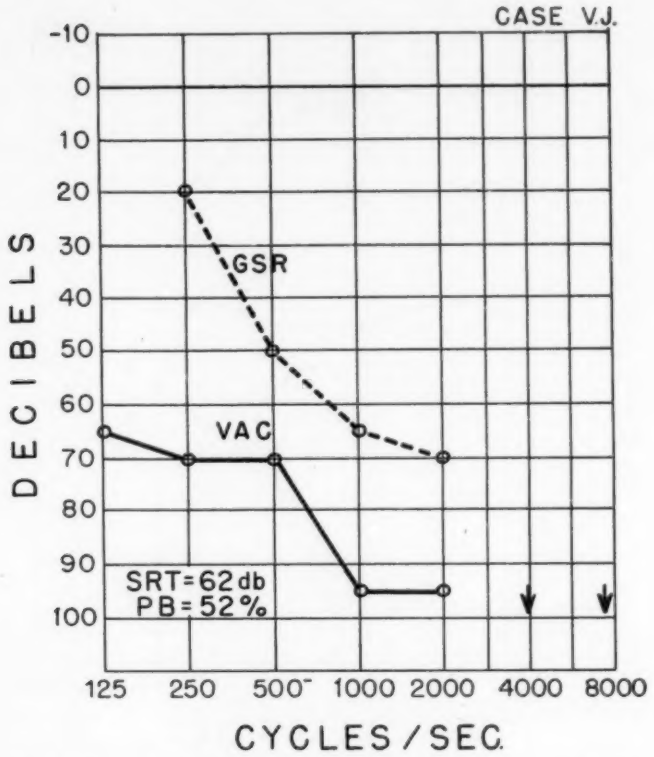

Fig. 7. Case V.J., Possible relationship between degree of recruitment and extent of overlay illustrated by smaller overlay at frequencies with greater preceptive involvement (500-2,000 cps). VAC = voluntary air-conduction.

non of recruitment probably causes stimuli to be perceived as relatively loud just above threshold. There may be complete recruitment at 4,000 cps in this case. An examination of his right ear thresholds (see Fig. 6-b, Case N.A.) suggests that the degree of recruitment influences the amount of functional overlay. At 2,000 cps the perceptive decrement is only 20 db, and while some limiting effects of recruitment appear to be present, the decrease in the extent of functional overlay is not so marked as it is at 4,000 cps. It is probable that a lesser degree of recruitment was present at 2,000 cps.

The next case illustrates a more complicated relationship in a 43-year-old white male janitor with a bilateral mixed perceptive hearing loss, chronic severe bilateral otitis externa, a history of suppurative otitis media, and a functional overlay. His hearing loss (see Fig. 7, Case V.J.)

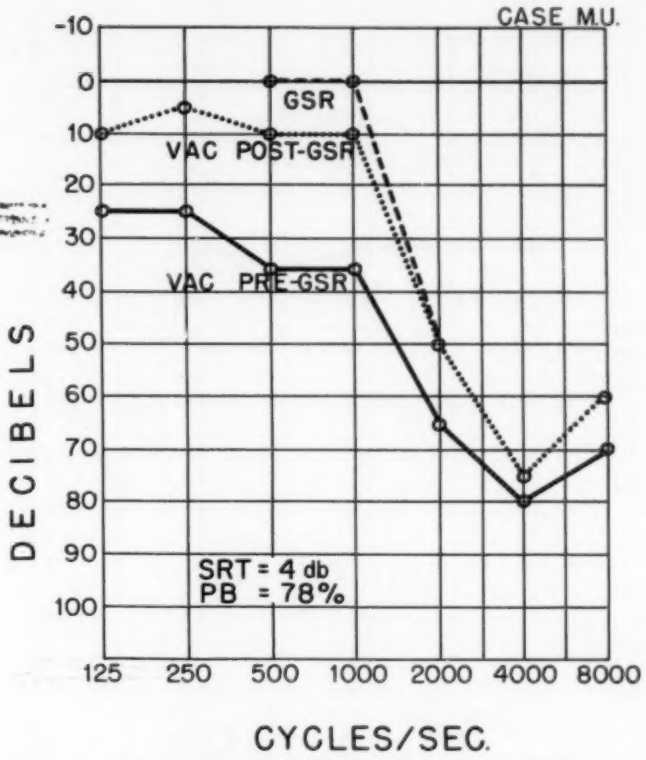

Fig. 8. Case M.U., Change in voluntary air-conduction (VAC) response after GSR and counseling illustrating small changes at higher frequencies.

is primarily perceptive in nature with a small conductive component. At 250 cps it can be seen that he has a 50 db overlay, while at higher frequencies the overlay is only half as great. This can probably be explained on the basis of greater perceptive involvement and greater recruitment at frequencies above 250 cps.

The next example demonstrates a change in voluntary response after counseling. The patient, a 45-year-old white male, gives a history of having been an aerial gunnery instructor for 18 months during World War II. Physical examination was essentially negative, and the final diagnosis was bilateral perceptive hearing loss due to acoustic trauma, with mild functional overlay. It can be seen (see Fig. 8, Case M.U.) that the post-GSR voluntary thresholds still reflect a small overlay at 500 and 1,000 cps, but the gap is closed completely at 2,000 cps where recruitment might have restricted the initial overlay. While the magni-

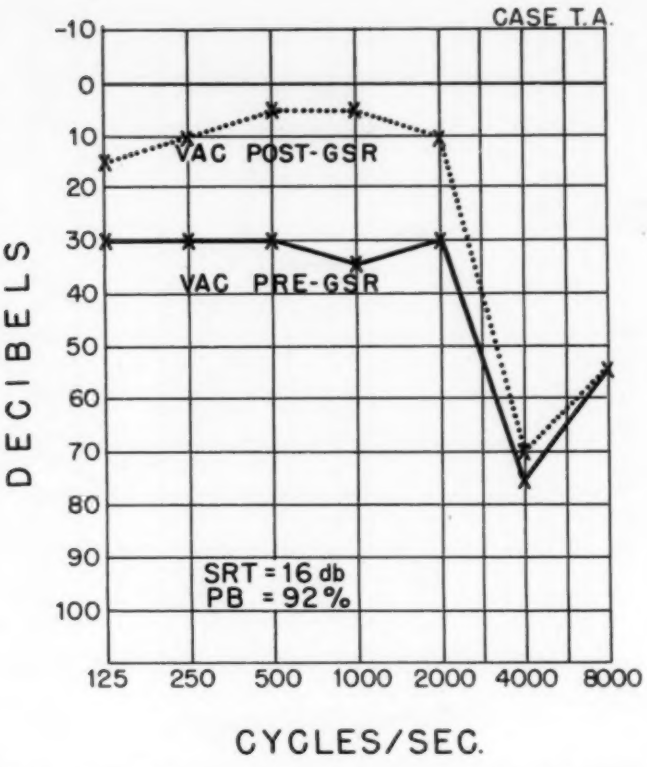

Fig. 9. Case T.A., Change in voluntary response after GSR illustrating close correspondence between pre- and post-GSR voluntary air-conduction (VAC) patterns at 4,000 and 8,000 cps where one might expect recruitment.

tude of the initial overlay in this case was relatively small at all frequencies tested, the least amount of overlay occurred at 2,000 and 4,000 cps—15 and 5 db respectively.

The stimulus situation of GSR audiometry occasionally results in a remission of functional symptoms. The following example illustrates this phenomenon in a patient after an unsuccessful conditioning attempt in GSR audiometry. The patient, a 36-year-old white male, dates the onset of his hearing loss to 1945, when he was blown out of a tank by an explosion which knocked him unconscious and left him with multiple shrapnel injuries. At the time of his last examination ENT findings were negative. It can be seen (see Fig. 9, Case T.A.) that at 4,000 and 8,000 cps, where one might expect some perceptive involvement, the

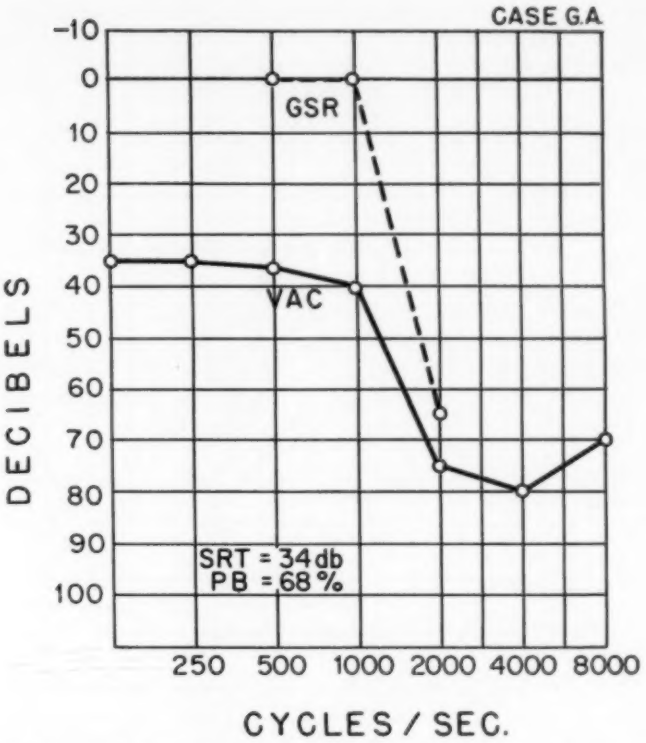

Fig. 10. Case G.A., Minimal overlay at 2,000 cps indicates presence of recruitment (see Fig. 11).

post-GSR curve corresponds very closely with the pre-GSR curve, a plausible result in the presence of recruitment.

The last example clearly illustrates the relationship between recruitment and the extent of functional overlay. The patient is a 39-year-old male who traces the onset of his loss to a gun muzzle explosion in 1945. Immediately following the explosion he complained of total deafness and severe tinnitus. Both symptoms gradually improved but still remain problems. The ENT examination was essentially negative. GSR findings for the right ear (see Fig. 10, Case G.A.) reveal normal hearing at 500 and 1,000 cps, but a 65 db loss at 2,000 cps. The overlay, then, is 35 and 40 db at 500 and 1,000 cps respectively, but only 10 db at 2,000 cps. After counseling the patient's voluntary thresholds improved to such an extent that a loudness matching procedure was thought feasible. The results of this test (see Fig. 11) indicate complete recruitment at 2,000 cps in the right ear. Essentially the same results as described above

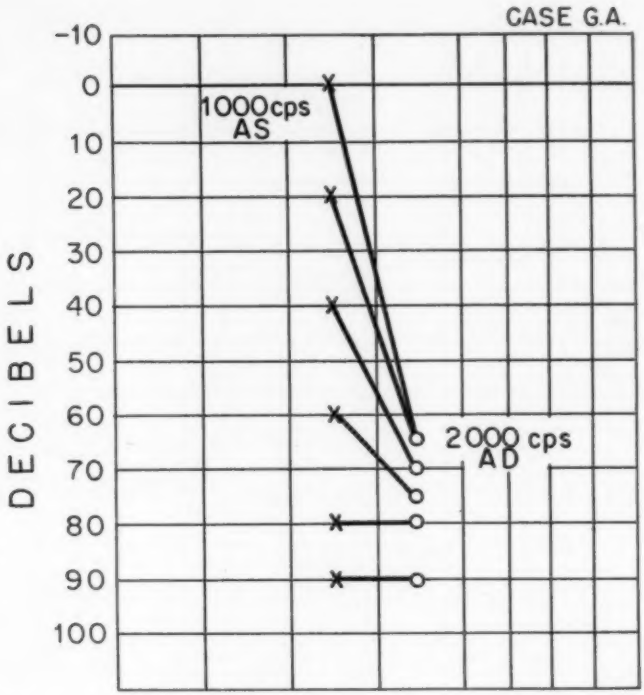

Fig. 11. Loudness matching results for Case G.A. Loudness matches were made between the left ear at 1,000 c.p.s. and the right ear at 2,000 cps.

were found for the left ear. These results appear to support the hypothesis that recruitment, and perhaps the degree of recruitment, plays a significant role in determining the extent of functional overlay.

CONCLUSIONS.

Several general statements may be made on the basis of the foregoing information:

1. Saucer-shaped audiograms can appear on a purely organic basis. It is not well established, furthermore, that the saucer audiogram is the most probable functional pattern in patients with no underlying organic impairment. It is our

experience that only a small percentage of functional hearing loss cases have saucer audiograms. We have also found that other clinical data can be used far more effectively in the detection of functional involvement.

2. There does not appear to be a single pure-tone threshold configuration characteristic of functional hearing loss. The diversity of pure-tone patterns observed in functional loss makes it difficult to identify functionality solely on the basis of pure-tone threshold configuration.

3. One of the primary limitations in magnitude of functional overlay appears to be the presence of recruitment. The cases presented in this article would seem to support this hypothesis. Further research in this area should produce a more definitive statement of the mechanisms involved.

BIBLIOGRAPHY.

1. Semenov, H.: Deafness of Psychic Origin and its Response to Narcosynthesis. *Trans. Amer. Acad. of Ophthalmol. and Otolaryngol.*, pp. 326-348, March-April, 1947.

2. Fournier, J. E.: The Detection of Auditory Malingering. Trans. *Beltone Instit. for Hearing Research*, No. 8:3-23, Feb., 1958. Translation of: Le Dépistage de la Simulation Auditive. *Exposés Annuels d'Oto-Rhino-Laryngol.*, pp. 107-126, Masson & Cie, Paris, 1956.

3. Doerfler, L.: Psychogenic Deafness and Its Detection. *Arch. Otol., Rhinol. and Laryngol.*, 60:1045-1048, Dec., 1951.

4. Johnson, K. O.; Work, W. P., and McCoy, G.: Functional Deafness. *Arch. Otol., Rhinol. and Laryngol.*, 65:154-170, March, 1956.

5. Carhart, R.: Audiometry in Diagnosis. The Laryngoscope, 68:253-279, March, 1958.

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SIXTH INTERNATIONAL CONGRESS ON DISEASES OF THE CHEST.

The Sixth International Congress on Diseases of the Chest will be held at the University of Vienna from August 29 to September 1, 1960.

STAPEDIOPLASTY.

A New Concept for Stapes Surgery.

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Louisville, Ky.

The recently revived technique for restoring functional stapes mobility in cases of clinical otosclerosis was directed initially toward breaking through the otosclerotic lesion.[1] While a significant number of patients have maintained a serviceable level of hearing following this procedure, initial failure to obtain improvement and subsequent refixation have continued to frustrate the efforts of the otological surgeons. Various modifications of technique have been evolved with the hope of increasing the percentage of lasting hearing improvements. These have included, *1.* perforation (fenestration) of the footplate[2]; *2.* vein graft and polyethylene insert after stapedectomy,[3] and *3.* removal of the stapes crura with fragmentation of the central area of the footplate and insertion of a tantalum pin between the incus and footplate to replace the crura.[4]

The third method, which was conceived by Schuknecht, seemed to be the most logical and least hazardous, but has the objection of having a foreign substance introduced into the middle ear. Schuknecht states, however, that his observations on this method extend over two years, and to date no clinical evidence of intolerance to tantalum has been noted.

It occurred to me that if a precise crurotomy technique could be developed, then one crus could be transposed to a fragmented footplate and a good functional result obtained. This would also bypass the otosclerotic lesion and possibly lessen the likelihood of refixation. Good long range results obtained by me with Fowler's technique[5] in some cases also suggested the feasibility of this new concept. Fowler stated that, in selected cases, the uninvolved posterior half of the

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footplate could be cracked off from the anterior otosclerotic portion after an anterior crurotomy had been performed. The patient then has a monocrural structure which, if unimpeded, provides a very satisfactory level of hearing.

The chief obstacle was the lack of adequate instrumentarium to carry out precise crurotomy technique. Pointed picks were

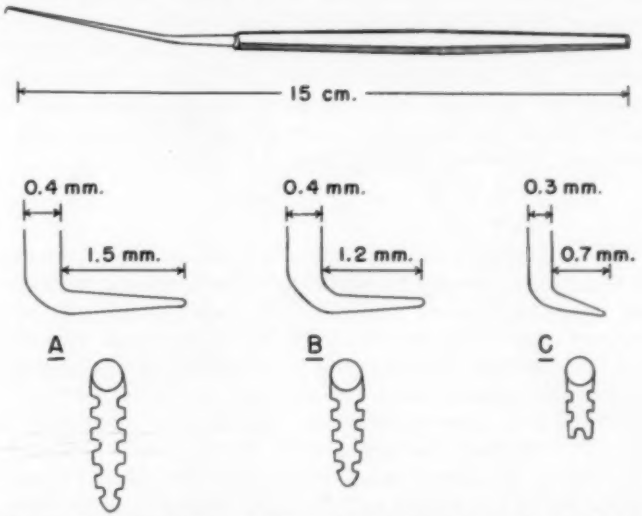

Fig. 1. The overall size and shape of a crurotomy saw are shown in the upper part of the illustration. A shows an enlarged side and top view of an anterior crurotomy saw with a long blade. B shows a slightly shorter one. C represents a posterior crurotomy saw. It is designed to cut chiefly from the end of the blade. The angle between the shaft and blade is about 100 degrees. This permits a slight "excavating" maneuver in separating the crus from the footplate in order to provide maximum length. The shaft of each saw is malleable so that it can be bent to the appropriate angle for each case. Available from V. Mueller & Co.

Before the saws are put away after use, each blade should be cleaned with a soap solution and very fine brush. Otherwise corrosion will occur and cold sterilization may not be effective. This cleaning should be done under magnification.

sometimes used for this purpose with success, but generally were not adequate because manipulation caused fractures of the crura where not desired. Scissors tended to twist and crack the other crus in the wrong place. After considerable technical experimentation, I was able to create microscopic

crurotomy saws (see Fig. 1). This made possible a precise cut across either one or both crura at the exact point desired.

If, on initial inspection and palpation of the stapes, it appears that the otosclerotic fixation involves only the anterior footplate area, and that the posterior portion will mobilize easily, then Fowler's technique can be tried. A good separation of the posterior mobile portion of the footplate from the fixed anterior portion should then provide a good hearing improvement. This method will be successful in only a relatively small number of cases. If it is not successful, then the surgery can be extended to the monocrural transposition technique. In cases with extensive vascular otosclerotic infiltration of the footplate, surgical manipulations should be minimal. These are best reserved for fenestration.

TECHNIQUE.

The preliminary medication, local anesthesia and elevation of the tympanomeatal flap to expose the tympanic cavity will not be detailed, since this phase of stapes surgery is fairly uniform as used by various surgeons. I use loupe magnification for the flap elevation and then use 16X Zeiss operating microscope magnification for the rest of the surgery. It is imperative to remove sufficient meatal bone to provide exposure of the posterior crural area, the stapedius tendon and the edge of the facial canal adjacent to the stapes. Careful inspection of the stapes and footplate should then be carried out. Any mucous membrane bands or webs which obstruct the view should be separated with an appropriate probe or hook.

If the central portion of the footplate appears somewhat bluish and thin, the prognosis of successful surgery is excellent. Gentle transincudal manipulation should be tried first. Occasionally this will free the footplate and provide a good functional result without further surgery. When the footplate is more rigidly fixed and the situation appears favorable for Fowler's technique, then this should be attempted with a crurotomy saw. If the posterior crural area is rigidly fixed, then the monocrural transposition technique can be carried out.

Steps of stapedioplasty (monocrural transposition):

1. The stapedius tendon is cut to provide better posterior crural exposure and maximum crural mobility.

2. An anterior crurotomy is started with a crurotomy saw (see Fig. 2). Delicate, meticulous handling of the saw is imperative if an inadvertent twist fracture of the crura in the

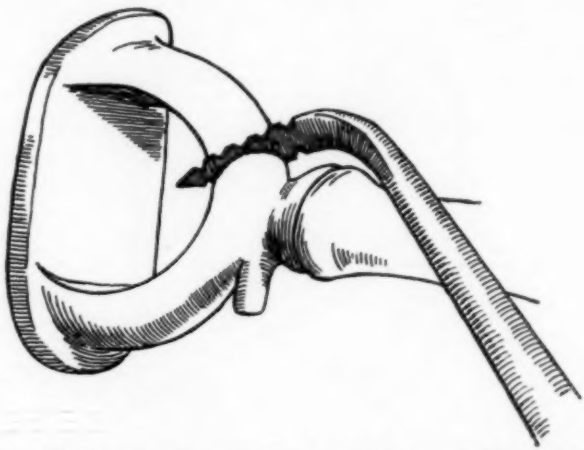

Fig. 2. The view of the stapes is represented somewhat more oblique than is true of the actual view obtained at surgery in order that the saw technique can be shown clearly. Otosclerotic lesions were not shown in these drawings. Whether the approach with the saw is from the superior or inferior aspect of the crus depends on the angle of the particular stapes and its relation to the promontory and facial nerve.

wrong place is to be avoided. The direction of the movements of the long axis of the anterior crurotomy saw should be oblique (45 degrees) to a plane passing through both crura. Direct side pressure (90 degrees) to the crural plane will frequently fracture the crura. In using the anterior saw there must be constant vigilance in order to avoid trauma to the facial nerve. In some instances the view of the anterior crus is obscured by the incus and capitulum. It is then necessary to do the crurotomy by touch. In one instance a short saw was used to approach the anterior crus from its under-

neath aspect. The shaft of each saw is malleable and can be angled as needed to fit the individual case.

3. Before the anterior crurotomy is completed, another type of saw is used to cut partly through the posterior crus *as close to the footplate as possible* (see Fig. 3). This saw approaches the posterior crus from the anterior-superior or anterior-inferior aspect. Cutting partly through the posterior crus at this time will provide a weak place in the posterior crus, and

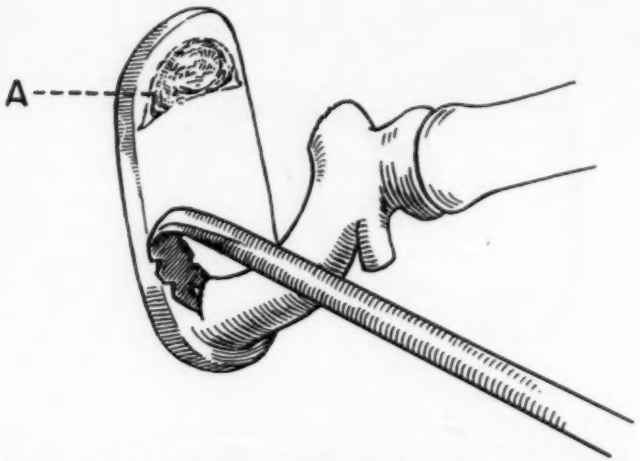

Fig. 3. Posterior crurotomy saw in place adjacent to the footplate. The approach is usually from the anterior superior aspect of the crus but for diagrammatic convenience it is shown on the inferior side. Note the cutting is accomplished chiefly by the end of the saw. A shows the stump of the anterior crus.

when the anterior crurotomy is completed any inadvertent twist of the body of the stapes will tend to crack the posterior crus at the footplate, thereby providing maximum length.

4. After the anterior crurotomy is completed, the remainder of the anterior crus distal to the cut is fractured and removed.

5. The posterior crurotomy is then completed with a saw and a right angle sharp probe. Intense, prying pressure with a probe behind the posterior crus attachment should be avoid-

ed, because this may fracture the crus a short distance away from the footplate rather than at the footplate.

6. The freed posterior crus is now reflected forward to expose the footplate (see Fig. 4).

7. Fragmentation of the footplate is now carried out (see Fig. 4). This is accomplished with a sharp probe and knife. The knife is not used to perforate but merely to scratch the footplate surface in a manner similar to that used in cutting glass. Separation of the footplate fragments is unavoidable,

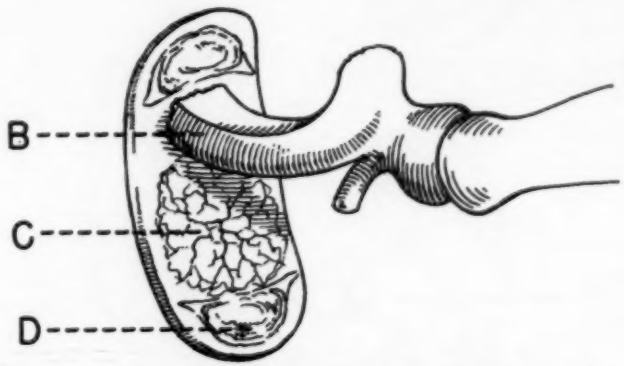

Fig. 4. B—Posterior crus reflected forward to expose footplate. C—Fragmented area in center of footplate. D—Site of posterior crus cut at or below footplate level.

and is followed by some escape of perilymph. Perforation or penetration into the vestibule is avoided. Suction directly on the footplate should now be avoided in order not to remove any excess perilymph. *Excessive* inward displacement of footplate fragments is inadvisable. A few small dehiscences may be desirable in order to provide more mobility of the fragments. Whether a dehiscence decreases the liklihood of refixation remains to be seen.

8. The posterior crus is now placed over the fragmented area at the point where it seems to establish the best possible contact with the footplate (see Fig. 5). In the event that contact is lacking, a minute piece of conchal cartilage might

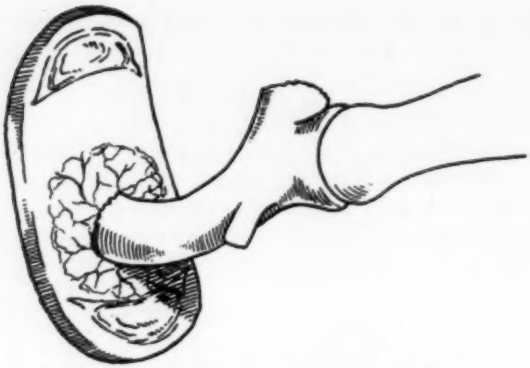

Fig. 5. Posterior crus transposed to fragmented footplate at point of optimum contact.

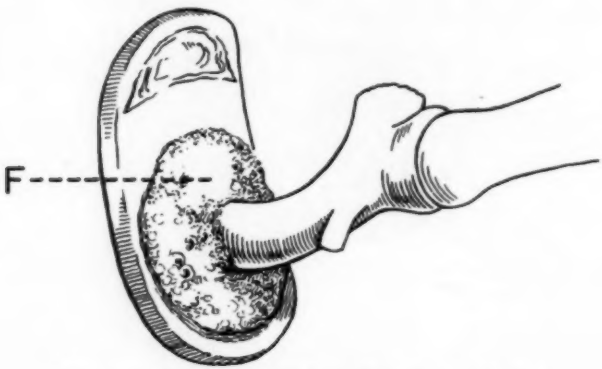

Fig. 6. F—Gelfoam placed around transposed crus.

be used to fill the gap. Meatal bone could also be used, but it might theoretically be more inclined to initiate osteogenetic activity.

9. Two or three minute pieces of gelfoam are placed around the transposed crus to hold it in place and seal the perilymph space (see Fig. 6).

10. The middle ear is then aspirated to remove any residual blood or fluid. The canal anesthesia solution frequently gravi-

tates into the middle ear and should be aspirated at intervals during as well as on completion of the operation. The round window membrane is not impermeable to topical anesthesia, and prolonged contact of the solution could conceivably penetrate the inner ear.

11. The tympanomeatal flap is placed back in position, and the incision covered with a few pieces of gelfoam.

If the posterior crus is involved in otosclerotic fixation and the anterior crus and adjacent footplate are not involved; then

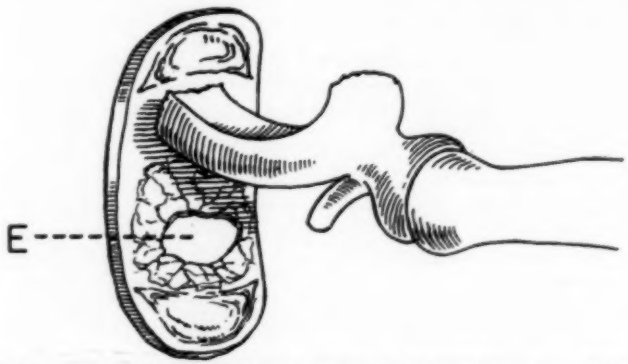

Fig. 7. E—Dehiscence created in center of fragmented area. Posterior crus to be placed over this footplate opening.

a posterior short crurotomy can be done and the anterior crus transposed to the fragmented footplate. This situation occurred in one case.

There are occasional instances in which the facial nerve is so close to the promontory that there is space for only the crura. The bony canal over the facial nerve at this point is frequently absent in such cases. It is impossible to view the footplate in such situation and no direct footplate surgery is possible.

RESULTS.

I have used the stapedioplasty (monocrural transposition)

technique on ten patients to date (cases 1-10, see Fig. 8), the first one having been performed in September, 1958. The lower end of the arrow shows the preoperative speech frequency pure tone average, and the arrow point indicates the optimum level attained after surgery, usually one or two weeks postoperative. It will be noted that the air-bone gap

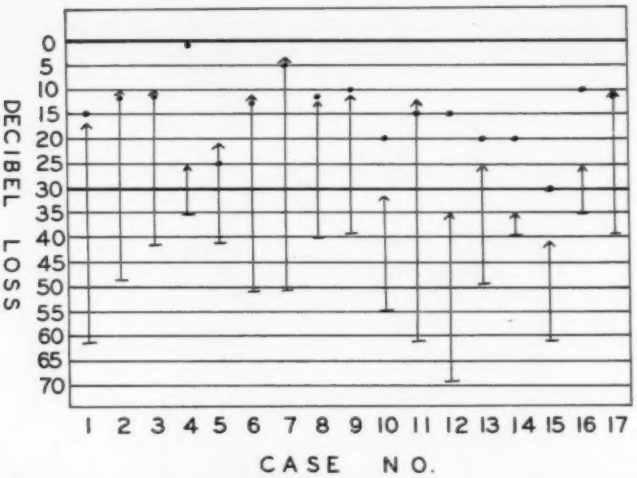

Fig. 8. This chart shows the pure tone threshold average for the 500, 1,000 and 2,000 frequencies on 17 most recent cases of stapes surgery. The solid dot represents the preoperative bone conduction threshold. The horizontal bar at the lower end of the vertical line indicates the preoperative air conduction threshold. The arrow point at the top of the line shows the best early postoperative air conduction threshold. This was usually obtained two or three weeks after surgery. Cases on which satisfactory monocrural transposition was accomplished are grouped in the first 10 shown. Other techniques are grouped in 11-17 as discussed under the section "Results."

has been eliminated in all cases except Nos. 4 and 8. In Case 4 the footplate was somewhat thick, even though it appeared somewhat bluish. This was one of the early cases, and subsequent experience would indicate that the amount of fragmentation accomplished was inadequate. If the improved level of hearing is not maintained, the operation can be revised, and the degree of fragmentation increased.

In the process of fragmenting the footplate, it is impossible,

with the present technique, to avoid creating a small dehiscence in addition to the minute punctures. What influence this may have on the permanency of fragment mobility remains to be determined. In reporting on their histological examination of a case of otosclerosis 15 months after stapes-mobilization operation, Altman and Basek[6] report as follows:

"The tear in the endosteum healed completely, and the defect in the footplate was filled with a layer of connective tissue. There is very little reaction and definitely no bony callus formation around the broken pieces of the footplate, and callus formation is also absent in the fractures through both stapedial crura. The latter consist of skein-like bone.

"The absence of labyrinthine reactive changes, particularly the absence of connective tissue proliferation around the bone chip sticking into the lumen of the vestibule, is noteworthy. It is, in all probability, the result of the prevention of a post-operative infection by tetracycline."

In view of this observation, the possibility that some dehiscence could increase the probability of permanency of mobility must be considered.

In Case 10 I purposely created a footplate opening in the area of fragmentation, and placed the transposed crus over this opening and packed gelfoam around the crus (see Fig. 7). There was considerable postoperative tinnitus, and the hearing test four days after surgery showed less air conduction improvement than has been observed in most of the other cases; in addition, there was a slight depression of bone conduction acuity for the higher frequencies.

The permanency of the results obtained by the monocrural transposition technique must await the passage of time. Case 1 has maintained a 20 db level at three months after surgery. Case 2 has a 15 db level at two months. The remaining cases have had only six weeks' or less follow-up at the time of writing.

In Cases 11 and 12 the nature of the pathology was such that the longest crus obtainable did not reach the footplate; in fact, in each instance only half a crus-length was available. A small piece of meatal bone was inserted between the end

of the crus and the footplate. Whether this bony insert will stimulate osteogenesis and refixation cannot be stated at present. Possibly a small piece of conchal cartilage would be preferable to bridge the gap. In Case 11 the functional result was satisfactory, but in Case 12 it was not.

I plan to revise Case 12, remove the remains of the stapes and create a new "stapes" out of conchal cartilage. It should be possible to cut such a structure under 16X magnification. A small hole near one end perpendicular to the long axis of the new stapes would permit it to be pushed onto the incus. This would prevent penetration of the "fabricated stapes" into the perilymphatic space of the vestibule. It is my feeling that, in instances where a crural transposition cannot be accomplished for technical reasons, autogenous tissues should, if possible, be used to establish functional ossicular continuity. The alternative to this is to use an insert such as tantalum or polyethylene.

In Cases 13 and 17 the entire stapes was easily mobilized, and the crura were not cut. In Case 14 there was considerable otosclerotic infiltration of the footplate and only minimal mobility was obtained. In Case 15 a monocrural transposition was planned, but an attempt to loosen the posterior crus from the footplate by prying behind the crus caused a fracture of this crus in its mid-portion. This made transposition impossible, and a Fowler type of operation was carried out. It was not possible to overcome fully the impedance to good mobility. In Case 16 a fair degree of preoperative ossicular function was present before surgery, and it was not deemed justifiable to use the crural transposition technique.

DISCUSSION.

When the entire stapes footplate cannot be easily mobilized, the ideal technique would seem to be one which would bypass the otosclerotic lesion and yet maintain some type of ossicular functional continuity with a mobile section of the footplate, without penetrating or disturbing to an appreciable degree the contents of the vestibule of the labyrinth. Fowler's technique was the first to be directed toward this objective; however, even in seemingly suitable situations, the area of the posterior

crus may be fixed, even though there is no gross evidence of otosclerotic fixation at this point on inspection.

In the technique described by Schuknecht, the central thin area of the footplate is fragmented, the stapes crura are removed and functional continuity established by the insertion of a fine tantalum wire between the incus and the fragmented footplate. Disturbance of the vestibular contents here is minimal, but the theoretical objection to the introduction of a foreign substance has been raised; however, as far as is known, tantalum is well tolerated in all tissues.

Shea has evolved a technique in which he removes a large segment of, or the entire footplate and both crura. He seals the oval window with a vein graft, and inserts a polyethylene tube between the incus and the invaginated vein graft. This obviously creates considerable disturbance of the vestibular contents. Long range tolerance of polyethylene exposed in the tympanic cavity in this manner remains to be determined. Wullstein has stated that, in his experience, plastic prostheses used in tympanoplasty surgery are eventually rejected by the tissues; however, the plastic material he used was not polyethylene.

The stapedioplasty technique described by the author employs the footplate fragmentation principle of Schuknecht, but instead of using a foreign substance for restoring functional continuity, utilizes precise crurotomy and monocrural transposition for re-establishing ossicular continuity. Hearing improvements obtained by this technique in a small series of cases have surpassed results obtained by previous methods in the author's experience.

An improvement in the means for fragmenting the footplate is needed. Further refinement of instrumentation is possible. If the mucous membrane on the footplate could be elevated before fragmentation is started, then the membrane could be rolled back on completion of the fragmentation and serve to seal the perilymph space. This might also lessen the tendency toward scar tissue formation around the footplate fragments. A microscopic knife and elevator are being developed for this purpose. An incision through the mucous

membrane between the crural sites with elevation away from this line should encounter very little bleeding.

Whether gelfoam is the best material to pack around the transposed crus is to be considered. Small pieces of tympanic mucosa from the promontory could be considered for this purpose. If the transposed crus maintains its new position, no packing at all may be needed.

If there is eventually any tendency toward refixation of the footplate fragments, creating a larger dehiscence and covering this opening before transposing the crus should be considered (see Fig. 7). A small piece of thin perichondrium could be easily obtained for this purpose and placed under the mucous membrane flap. A vein graft as used by Shea could be similarly utilized; however, obtaining a vein graft adds to the complexity of the operation.

It should be pointed out that if crural transposition is used as a salvage procedure after inadvertent fracture at points other than those indicated in the section on technique, then optimum results cannot be anticipated. If, after initial inspection of the stapes, monocrural transposition seems to be indicated as previously discussed, then the technique described can be carried out precisely, and a high degree of predictability of hearing improvement should be possible.

SUMMARY.

1. A new technique for stapes surgery in clinical otosclerosis is described. It incorporates the following features:

a. The new conduction pathway bypasses the otosclerotic lesion.

b. Re-establishes functional ossicular continuity by monocrural transposition.

2. The hearing improvement to be anticipated seems to be highly predictable in properly selected cases as determined by initial inspection and palpation of the stapes at the time of surgery.

BIBLIOGRAPHY.

1. Rosen, S.: Palpation of the Stapes for Fixation. *Arch. Otolaryngol.*, 56:610-615, 1952.

2. Rosen, S.: Fenestra Ovalis for Otosclerotic Deafness. *Arch. Otolaryngol.*, 64:227-237, 1956.

3. Shea, John, Jr.: Discussion of Stapes Mobilization Symposium. *Trans. Amer. Laryngol., Rhinol. and Otol. Soc.*, 1958.

4. Schuknecht, Harold: Discussion of Stapes Mobilization Symposium. *Trans. Amer. Laryngol., Rhinol. and Otol. Soc.*, 1958.

5. Fowler, E. P., Jr.: Anatomic Factors in Stapes Mobilization Operations. *Arch. Otolaryngol.*, 63:589, June, 1956.

6. Altman, Franz, and Basek, Milos: Histological Examination of a Case of Otosclerosis 15 Months After Stapes Mobilization Operation. *Arch. Otolaryngol.*, 68:314-324, Sept., 1958.

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THE BIOCENOLOGIC APPROACH TO BLACK HAIRY TONGUE WITH NEW CONCEPTS OF ITS TREATMENT.*

Secondary Mycosis Linguae of Pathologic Significance.

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The present study of nine case reports and analysis of 118 related cases of "coated" tongue varying from white, patchy to yellow-brown, is the culmination of three years consideration of the problem of the etiology and treatment of *black hairy tongue,* and a search for a unifying concept that would explain all of the various manifestations and apparently conflicting ideas about the disease. The author's own concepts have been variously hit-and-miss, until early 1958 when fruitful crystallization of the parameters appeared in two timely articles, one by Winer[1] and the other by Makower.[2] The latter is the more valuable of the two, with the discussion of *biocenosis* which, for the present author, contains the unifying concept so long sought. Both articles will be considered in more detail further on in this paper. Other acute and chronic diseases, often involving coating of the tongue, are not related to the entity under discussion and will not be considered here. These include: leucoplakia, phenol poisoning, scarlatina, acute tonsillitis, typhoid fever, typhus, measles, and Vincent's angina. First, it would be valuable to mention what has been done up to now in the attempt to analyze the entity of *black hairy tongue.*

HISTORICAL.

Heidingsfeld[3] in 1910 was the first to review the literature on the subject, and he credits Rayer with the original description of the disease in 1835. At this time (1910) the etiology was considered of two types: a *true* hairy tongue

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that originated as an anomaly of development from embryonic life and developed later, possibly confined to definite areas of the tongue dorsum, and a *false* hairy tongue that owed its origin to local irritation, infection, tobacco, antisepsis or syphilis. In his Case 1, Heidingsfeld described filaments projecting from the dorsum of the tongue which could often be traced to an origin from papilla-like sites in the stratum corneum. These filaments often preserve a feather-like appearance. Another characteristic is the formation of abnormal papilla-like bodies, or epithelial nests, which he called "epithelial founts." A more accurate description of the pathology is that of Laskaris and Curtis[4] in 1949, who describe filiform papillae which have the superficial appearance of hairs. One has only to recall their ectodermal origin. The feathery nature of the filaments is important in serving to trap micro-organisms, food particles and tobacco stains, much as the baleen of the right whale traps crustaceans for its nourishment. This aspect will be considered in more detail later.

Pillsbury[5] in 1956, described the clinical picture as being made up of brown to black pseudopapillae confined to the posterior half of the tongue dorsum. He considered the etiology to be as yet unproven.

Kleinert[6] in 1946, thought the etiology was due to excess tobacco, and was unable to effect a cure with large doses of penicillin.

Layne and Watson[7] in 1941, caused a deficiency of nicotinic acid in dogs and developed lesions on the tongue similar to black tongue in the human. They also postulated a relation of black hairy tongue to human pellagra.

Weidman[8] in 1928, reviewed the literature and found one case associated with diabetes. It is noteworthy that all the teeth had been extracted years prior to development of the black tongue in this case.

In a survey of the literature in 1949 Laskaris and Curtis[9] found a more or less definite floral association in BHT: micrococci, alpha hemolytic streptococci, gram negative diplococci, leptotrichia, monilia, and actinomyces-like organisms, filiform

bacilli; the proportion varied with the case. They conclude, with serious doubt, that there is a parasitic etiology of the condition. Some, or perhaps most, of the bacilli and acinomyces-like organisms were found to be *lactobacilli;* in fact, organisms in two out of three cases studied by these authors referable to *lactobacillus acidophilus* were found to be implanted in the hairy tongue filaments. The rather consistent presence of aciduric-type organisms indicates they have supplemented normal tongue flora as a result of increased acidity of salivary secretions; their fermentative acidic activities insure a continuous chemical irritation, as noted by Swinburne.[10]

MODERN EVIDENCE.

Winer[11] describes the filiform papillae as being grouped in parallel rows across the tongue and as being most numerous of all papillae. The epithelial cells covering these form a hollow-shaped cap which normally undergo hyaline degeneration and are cast off. In pathologic conditions in man they develop heavy keratin tips, while in animals they are normally so thickly covered with keratin that they add a rasp-like appearance or "feel" to the tongue surface. He considers the etiology as made up of various bacteria, fungi, antibiotics, mouthwashes, and nicotinic acid deficiency. The pigment is definitely *not* melanin. The BHT contains increased keratin with an increased number of *monilia*. Through excess acidity keratin is not shed. Conversely there is excess shedding seen in the geographic tongue. This latter is frequently inhibited with dilute hydrochloric acidification. As to the pathology, he found that oral antibiotics may produce epithelial hyperplasia of the filiform papillae, while parenteral penicillin produces a proliferation of the capillary endothelium, an eosinophilic infiltration in the perivascular area of the papillary cutis, and is a pseudokeratosis without hypertrophy of the stratum corneum. He also noted that an observed vacuolation of the upper epithelium in sections of BHT was similar to that found in warts. He thereupon used a keratolytic, podophyllin locally, and in two weeks was able to control the case described in his interesting article. He advocated, in addition, oral troches of vitamin A to inhibit recurrence of

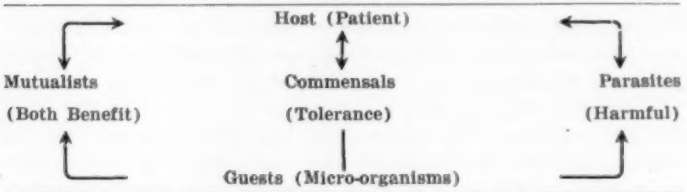
the keratosis. Montagna[12] in 1956, reported on the enzymatic hydrolysis by crop juices of ingested keratin in the hawk and vulture. This observation will have bearing in the discussion later involving *biocenosis.*

This brings the discussion up to the present time, and for the most part authors who have written on the subject have limited their presentations to the tongue alone, forgetting that the part is not the whole, and not seeing the tree for the moss, as it were. For the fully developed picture of BHT resembles nothing so much as a dense growth of moss on a tree trunk. What is more important though, is the fact that the patient himself is directly concerned about his state of nutrition and resistance, for example. Not just one isolated tissue or organ is to be considered in BHT, but the entire patient himself. The general tendency in medicine today is to treat symptoms and overlook the needs, pathology and treatment of the whole patient, despite the warnings of advanced medical circles. The concept of the whole patient must be extended to his family, neighborhood, community, region and finally to the nation; yes, and to the world as a unit. Herein lies the proposed value of the concept of *biocenosis* as noted by Makower[13] in 1958: coming from the Greek, *koinos,* meaning common or that pertaining to the community, population, or public. In more specific terms, it means a biologic community or population and the relationships of all members, both *macro* and *microscopic.* The term is similar to *ecology,* but this latter has a more limited meaning derived from the Greek, *oikos,* house. The term *symbiosis,* coined by deBary[14] in 1879, has come to mean predominately mutually useful associations, and Makower, therefore, uses the term *mutualism* for these. Since micro-organisms play a major part in the syndrome of BHT, their relationships as a *microbiocenologic* population on the mucous membrane and epithelium of the upper respiratory and alimentary tract must be considered here.

He notes three types of such relationships between the patient (host) and micro-organisms (guests) as follows (see Chart): *1.* When the relation between the two results in damage to the host, or in disease, it is known as *parasitism*; *2.* if it results in tolerance, *commensal*; and *3.* if both derive

benefit, *mutual*. One may verge gradually over a period of time into another; when antibiotics or other agents destroy the normal flora of the mouth, for instance, there results in the low resistance patient, or in the young or very elderly debilitated one, the acute infection known as "thrush," a parasitism. If, in the more resistant individual and in one in a relatively good state of nutrition, the antibiotics merely destroy the sensitive micro-organisms, leaving the resistant ones to develop and eventually "overgrow," then a tolerance is often noted and the commensal relation is seen. One often sees cases of "thrush" in children and one or more phases of BHT or other evidence of moniliasis, such as vaginitis or

CHART I.



Adapted from Makower,[2] 1958.

external otitis, in the adults of the family group. Frequently seen is external otitis with a concomitant fungus of the tongue in the same individual.

Many patients have given the history of antibiotics several years prior to having the BHT or milder modifications thereof uncovered during routine mouth and throat examinations, and they have, until then, been unaware they had a tongue lesion. Upon careful questioning they nearly always admit having a fetid breath or a "bad" taste in the mouth. Such lesions again are frequently noted by patients themselves, and only after careful recollection can they remember ever having had prior antibiotics. Only the most hardy of patients have no symptoms with their BHT; the usual finding is that mild to more serious pathology and localizing symptoms are presented. The number of invading organisms, or rather the number of resistant ones, which seem to be on the increase

with abuse of antibiotics, the immunity state and the nutritional balance of the individual determines the degree of involvement of the epitheliums mentioned above.

Sometimes fatal extensions, from pulmonary mycoses to the meninges and encephalon itself, are known to have occurred.[15] These variations (see bacteriology) determine the color of the tongue, whether white and patchy, or more diffusely yellow-brown to the diagnostic mossy BHT. Along with this lingual picture patches of keratotic vegetations have been found in the pyriform sinuses and even in the nasopharynx. BHT is not usually found associated with debilitating disease, such as diabetes or cancer. Antibiotics are the major causes of the syndrome, because of the injudicious use and abuse of their known usefulness; in fact, antibiotics themselves may be considered the product of a microbiocenotic relationship, that of antagonistic activities developed in other biocenotic communities, whether in the culture vat or in the human body.[16,17] One might, therefore, extrapolate backwards in time and speculate from Uri's finding that skin and hair fungi are able to produce antibiotics, that the etiology of BHT had a basis from the beginning of its description in 1835 (Rayer) in antibiotic activity, long before even the discoveries of Flemming. In a word, BHT nowadays may be considered a truly "iatrogenic" disease. Finland[18] notes that large doses of tetracyclines may also give rise to sore tongue or *black tongue*, cheilosis and rectal irritation in some patients.

The possibility of *mutations* in microbes as a result of acquiring resistance to antibiotics is a question only further research can elucidate. Mutation as a result of "fallout" from atomic and nuclear weapons testing is a definite possibility.

The public health aspects of antibiotic abuse are illustrated by the A.M.A. Committee on Medicolegal Problems,[19] which reviewed a report in *Veterinary Medicine*, which states that about 6 per cent to 12 per cent of milk reaching the consumer contains penicillin ranging from 2 to 80 units per glass. Many of my patients give a history of copious milk consumption, and not a few at one time or another during the winter

months have reported that their milk had a bitter or "off" taste.

Many patients volunteered the information that emotional strains or nervousness seemed to cause more "acidity" in the mouth and upper gastrointestinal tract. The psychoneurotic factors as one of many causes of BHT are interesting, but not conclusive.

METHODS OF TREATMENT AND MATERIALS.

Color Photography—In the present study only the more obvious BHT patients had their tongues photographed before and after treatment. One case of submandibular abscess, an extension from BHT, is presented in the color insert to this paper, and three photomicrographs of biopsy sections from the tongue.

Mechanical and Chemical—At first the usual monilicides, such as gentian violet or crystal violet, were tried as topical applications on the tongue, and then as a 1:100,000 mouth wash or gargle. In the acute "thrush" phase this would be relatively effective when Nystatin® was used concurrently; but at the time of application to a yellow-brown or black tongue, it was noted that very seldom did it penetrate below the protective mucoid blanket, and as a result the condition persisted. Mechanical scraping with a spoon-type curette was next tried with more success in directly reaching the tongue surface; next, a ball of gauze held with a Kelly forceps seemed to aid in removal of the slick blanket of mucus, but with all this mechanical effort the average time needed to clear up the surface of the tongue to a normal reaction and appearance was about two months, varying from two weeks to almost two years in one case. Often the condition would reappear, usually after a round or two of penicillin or other antibiotics. That the pioneer spirit is not yet dead is evidenced by the acumen of one patient in using the old corn cob to scrub the dorsum of the tongue, and when that began to hurt he switched to "sheeting" or burlap in six inch squares. This would peel the coating and "hairs" off like the layers of an onion. Lastly, the best effect could be obtained by a No. 10 blade held in the Kelly forceps and shaved or scraped

across the tongue. Persistence in cleaning is a definite requirement for success, both for the physician and patient. The latter was often instructed to use gauze 4x4s as an aid at home.

After the association of acidophilic bacteria and fungi was noted, strips of pH paper, known as pHydrion strips, and obtained from Micro Essential Laboratories, Brooklyn 10, New York, with a range of 4-9, were used on the tongue when the patient was first seen, and during the phase of treatment. The environment was definitely acid, pH 5 or 6; while normal controls were found to be consistently alkaline or neutral. During treatment with pancreatic enzyme therapy the reaction was also alkaline. It is interesting that confirmation of this pH factor as influencing the presence or absence of fungi was found in a recent communication by Yannoulis in 1957.[20] He was interested in the pH found in laryngeal papilomatosis and found *Monilia* most frequently with an acid pH. The lower the pH the more resistant was the fungal condition to treatment. The present writer has noted a tendency to this resistance of the lingual cenosis to treatment with low pH, but no definite conclusions can be reached at this time.

Among the various chemicals used in the many attempts to clear up lingual coating were: 3 per cent gentian violet, Larylgan,® Desenex,® Decapryn,® tincture of myrrh, and Cetylcide® in a 1:4 dilution with water.

More recently the alkaline mouthwash Cepacol® has been tried with good results in keeping the reaction in the mouth at a more favorable pH. It is also helpful in combatting the bitter, musky taste of the pancreatin powder. Plain baking soda has also been advocated as adjunct in brushing the teeth and dorsum of the tongue and keeping the reaction at an alkaline pH.

Bacteriology—The mouth, according to one observer, is the "dirtiest orifice of the human body." The contrast to the relative case with which monilia may be isolated from the vagina, the abundance and variety of resistant organisms in the oral epithelium makes isolation of the offending fungus relatively more difficult. Fungi are omnipresent here and

appear often in normal mouth cultures, but it is in the pathogenic states induced by antibiotics that they assume medical importance.

From the evidence to be presented in the case reports, the previous association of *Candida* and other fungi as well is accepted, but emphasis is on the secondary and parasitic nature of the infection. The baleen nature of the keratinized filaments is again recalled; however, in three of the nine proven cases of BHT *Geotrichum* was isolated. This fungus has a clinical picture, orally, almost identical with the oral lesions of *Candida;* in fact, culturing is the *only* way to differentiate between the two, as the treatment so far advocated has been very different for each.[21] This is contrary to a recent editorial comment which stated: ". . . it is quite evident that secondary infections due to fungi (particularly the monilia) are infrequent and do not constitute a serious problem with antibiotic therapy."[22] By using the Hyland Laboratories blood agar and Sabouraud's dextrose agar in the plastic plates or petri dishes, the following micro-organisms were isolated: diphtheroids, aerobic gram positive spore-forming bacilli, *Aero-bacter-Klebsiellas,* alpha streptococci, flavo-bacteria, *Cladosporium* sp., *Verticillium* sp., *Penicillium* sp., *Rhodotorula* sp., gram negative cocci and diplococci resembling pigmented *Neisseria* sp., *Candida* sp. not *albicans, Candida albicans,* and *Geotrichum.* Much of the color, therefore, could come from the various color formers here mentioned, in addition to decaying food particles, tobacco stains and other food colors, *e.g.,* licorice, blackberries, nuts, paregoric and iron compounds.

In order to be certain that, in taking cultures from patients, room contaminants were not grown and identified as coming from the tongue, several blood agar and Sabouraud's plates were exposed for 24 hours in the main treatment rooms, then placed in the incubator. All fungi grown were identified as non-pathogenic non-spore formers. Cultures were, at the same time, taken from normal appearing mouths and tongues, but no fungi were grown. An occasional yeast, not *Candida,* was isolated, as were normal mouth organisms.

Pathology—The Martin core hand drill was used to obtain

the specimens. Where possible stains for fungi were done in addition to the routine H&E procedure. One pathologist agreed with the diagnosis of BHT; the other deemed from the microscopic picture for simplicity's sake and consistency, that the specimen was a squamous papilloma. Chronic inflammation was present in both; in Case 7, the rete pegs were more irregular in their configuration. In the photomicrographs in the insert, the hyperkeratotic "caps" are easily seen, in spite of their somewhat tangential sectioning during processing. The "baleen" nature is seen to be due to the unshed keratinized filaments. The capture of these processes of debris and bacterial-fungal colonies is illustrated. This fundamental picture is no different than that found in the Pillsbury textbook and the "Atlas" of Arthur C. Allen. The biopsy site is generally in the midline of the posterior half of the tongue and anterior to the circumvallate papillae as the tip and edge or border of the tongue is not involved in this *biocenosis.*

Enzymes and Supportive Therapy—The pancreatin used in this study is the whole pancreas which had been defatted, completely dehydrated, and then powdered. It is about six times the U. S. P. or N. F. potency. When the patient was first seen and found to have one of the "coats" of the BHT syndrome, a pH determination was made; and usually the reaction was found on the acid side of the pH scale. A BA plate and Sabouraud plate were then planted with tongue scrapings. Next, the tongue was generously powdered with pancreatin which was allowed to remain on the tongue from five to fifteen minutes, the patient being instructed not to swallow while the powder is in the mouth. After spitting out the accumulated saliva, the affected area of the tongue was then shaved or scraped with the No. 10 blade held firmly with a Kelly curved forceps. It was found that the pancreatin had softened, and dissolved the mucoid blanket and filiform papillae involved. Lastly, the tongue was generously swabbed with a crystal violet aqueous solution; the patient was instructed how to clean his tongue at home and warned not to use the stain over five days at a time without resting several days to prevent reactions. Vigorous brushing of the dorsum of the tongue as often as practicable was advised.

Several patients volunteered the information that the taste of pancreatic powder, while not too pleasant and of a strong "musky" scent, was nevertheless much more palatable than Nystatin.®

CASE REPORTS.

1. Mr. R.G.B. was a 45-year-old carpenter, who sustained a basal skull fracture without loss of consciousness when he fell backwards from a six-foot ladder. While being treated in the hospital for a fracture through the right temporal bone involving the bony external canal, he developed BHT from extensive penicillin prophylaxis. A multitude of various products were tried, starting with gentian violet locally, and almost daily scraping with the large ethmoid curette. At times, in despair, he would consult with another physician, who prescribed antibiotic troches, which made the condition worse. He noted that after eating frozen chicken the condition which had almost cleared, would again become as extensive as formerly (see Fig. 1). A foul odor and taste was noted by the patient. The pH of the tongue was 5.

Several cultures of the tongue scrapings were necessary to isolate yeasts and fungi: *Cladosporium* and *Verticillium*, which at first was thought to be *Geotrichum*. Quite by chance he recently had several infected teeth removed by his dentist, and gum treatment for the remaining; the BHT persisted. Then on the advice of his dentist as a tartar control measure, he started using a proprietary tooth powder containing papain, and in one week the tongue lesion had disappeared. When he did not use the powder the coating returned, so he was put on Pancreatin powder charts three times a day and instructed to brush the tongue vigorously after 15 minutes. Since then the pH has remained neutral or slightly alkaline and the tongue remains clean (see Fig. 2). He also was placed on Entozyme® tablets t.i.d., Allbee with C® capsules p.c., and remains free of coating at the present writing.

2. Mr. R.T., a 76-year-old retired farmer, was routinely examined for epistaxis and found to have Rendu-Osler's disease. In addition the tongue was "mossy" (see Fig. 3), and he recalled after a few minutes having a series of penicillin shots for a "cold" about two years previously, but stated that he was in no discomfort. He refused biopsy and treatment but did permit culture of tongue scrapings. This grew out no fungi, but did reveal numerous yeasts, not *Candida*, micrococci, alpha-streptococci, diphtheroids and unidentified pigmented gram positive rods.

3. Mrs. P.C. was a 48-year-old accident-prone housewife with numerous complaints, who had received the gamut of antibiotics and sulfas for various auto accidents and a recent accidental bullet wound of the forehead. The missile grazed the skin and bone while she was cleaning a .22 caliber pistol. When she came in complaining of a sore throat, and wanting more penicillin for control, examination of the tongue revealed BHT (see Fig. 4). Cultures of the tongue showed non-hemolytic streptococci and diphtheroids. There were a variety of fungi present: *Cladosporium*, yeast, not *Candida*, and one colony of *Aspergillus*. She has done well on pancreatin and supportive vitamins. The tongue remains free of coating.

4. Mr. R.T., a 45-year-old chicken farmer from North Georgia was the most interesting and conscientious of all the patients. He recalled having a series of penicillin shots for a cold approximately four years prior to examination (see Fig. 5). During the interim he carried out his usual duties with his chicken farm, which involved dumping 500 lbs. of an

Fig. 1. The tongue of case 1. which had persisted for two and one-half years showing BHT.

Fig. 2. Same tongue as in Fig. 1 showing result after one week of pancreatin powder and vigorous scrubbing with toothbrush.

Fig. 3. Tongue of Case 2. showing the "mossy" appearance.

Fig. 4. *Aspergillus* was one of several fungi isolated in Case 3.

Fig. 5. Tongue of Case 4. from which *Geotrichum* was isolated.

Fig. 6a. Biopsy section of Case 4. (low power) showing clumps of bacteria and debris. Filiform hyperkeratosis is well illustrated.

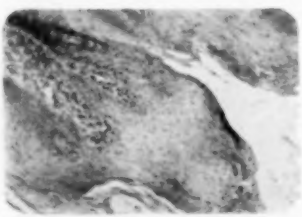


Fig. 6b. Higher power of section of hyperkeratotic epithelium. The accumulation of debris in the crypts favors growth of resistant organisms and the various fungi; these cannot be reached by ordinary solutions, but pancreatin being a keratolytic, enables various surface tension lowering agents to reach the source of the biocenologic growth.

Fig. 7. Tongue of Case 5. *Geotrichum* isolated from scrapings. Black "hairs" are in mid line posteriorly.

Fig. 8. The yellow-brown of case 6. *Geotrichum* was isolated.

Fig. 9. BHT that had persisted in Case 7 for at least six months. She was unable to protrude the tongue any farther, (See Fig. 10.)

Fig. 10. Same Case 7 showing left submandibular swelling in addition to fatty tissue normally present in this obese individual.

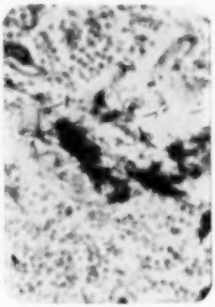


Fig. 11. Biopsy of tongue in Case 7, (oil immersion) showing clusters of small bodies which could not be distinguished from certain fungi. Her "coating" had disappeared in ten days.

Fig. 12 BHT of Case 8. A yeast and a *Cladosporium* sp. was isolated from tongue scrapings. The uveitis did not clear up until the tongue lesion was under control.

Fig. 13. Case 9. This was the most markedly black tongue of the series. A *Candida* sp. not *albicans* was isolated from the scrapings. He refused treatment and biopsy.

antibiotic feed at suitable intervals into the feeding troughs. Each time he would be engulfed in a cloud of feed dust and antibiotic powder. It is presumed that this was the cause of his BHT, constant daily exposure to small quantities of tetracycline and other antibiotics. Instead of buying an expensive tongue scraper at the local surgical house, he hit on the idea of using the old fashioned corn cob. This proved most helpful in cleaning the dorsum of the tongue, and when it became too raw, he switched to burlap or "sheeting" cut into 6-inch squares. This would enable him to peel the coating off like the layers of an onion. This procedure would fairly well control the lesion, but when, within the past month, pancreatin powder was advocated, his tongue cleared within one week. Cultures of the tongue showed *Geotrichum*. He consented to a biopsy of the tongue (see Fig. 6).

5. Mr. W.B., a 77-year-old business man, came in with a "bad cold" of many months' duration. Examination revealed an allergic rhinitis and BHT see Fig. 7). He gave a history of antibiotics eight months prior for the beginning of this "cold." Tongue cultures revealed *Geotrichum*. He has cleared up within ten days of supportive vitamins, Allbee with C,® Entozyme,® and Dimetane,® for control of his rhinitis. He denied prior history of hay-fever, and nasal smear showed up over 50 per cent eosinophiles.

6. Mr. B.A., a 24-year-old druggist, had received a series of antibiotics for an acute upper respiratory infection about six months prior to examination. During the ensuing months he noted a foul taste in his mouth and a constant "nagging" ache in his throat. The tongue was coated (see Fig. 8), and in less than a week, treatment with pancreatin powder t.i.d. on the tongue for 15 minutes, the lesion was gone and the tongue was normal. *Geotrichum* was isolated in cultures. Reaction of the mouth saliva was pH 5 prior to therapy, and when the tongue had returned to normal, reaction was neutral. He remains well with supportive B complex vitamins and occasional use of pancreatin powder.

7. Mrs. M.P., a 34-year-old nursemaid, began noting an intermittent left submaxillary swelling following eating, and a very "bad taste" to her mouth. She consulted several physicians, who prescribed various antibiotics, but with no alleviation of her complaints. When she was seen in the office on August 8, 1958, she had a hard tender mass in the submental region and point tenderness in and around the left submaxillary gland (see Figs. 9, 10). The tongue was definitely BHT. There was marked trismus, odyn- and dysphagia. The sublingual swelling limited her speech, and nourishment was chiefly liquids. Oral temperature was 102° F. She was accordingly admitted to the hospital with a tentative diagnosis of Vincent's angina secondary to abscess of the left submaxillary gland and BHT. Smear from the mouth did not reveal any fuso-spirochetal organisms and culture eventually grew out a hemolytic staphylococcus aureus. No fungi were noted. A biopsy was advised and agreed to (see Fig. 11). She was given daily I.M. one gram of Kanamycin for six days, and hourly hot saline irrigations sublingually and externally to the submandibular region. On the sixth hospital day the left submaxillary duct was catheterized after oral and external X-rays failed to show a salivary stone, and 0.3 cc. of iodized oil injected into Wharton's duct. The X-rays revealed a normal sialogram without any dilatation of the smaller ducts in the gland itself. X-rays of her teeth were negative. The tissue surrounding the duct was thickened and eventually with all her treatment the abscess opened just below the papilla of the orifice, and discharged about a cupful of foul-tasting pus. The best interpretation of the facts seems to be that the left submaxillary gland was not involved, but the surrounding lingual tissue was secondarily involved from the infestation of her tongue. The pH during the period of BHT was 5; after instituting pancreatin powder method and vigorous brushing of the tongue and

scraping with the No. 10 blade, pH was neutral, and since recovery of normal tongue appearance, pH has remained neutral.

8. Mr. E.D.L., a 54-year-old street sweeper was seen the fourth day after a severe injury to the left eye, caused by a tree branch being deflected by the windshield of the sweeper striking a deep gash in the cornea. He had been treated by another physician for the previous four days with antibiotics but no mydriatics. A widespread ulceration of his cornea was found, associated with a traumatic iritis. *Aerobacter* sp. sensitive only to chloramphenicol was cultured from the ulcer. He was responding poorly to standard treatment and was hospitalized for I.V. typhoid therapy. During this admission it was discovered that he had BHT (see Fig. 12), which grew out a yeast and a *Cladosperium* sp. Pancreatic powder therapy was then instituted and his vitamin intake increased. It was noted that his ocular disease did not improve until the tongue lesion was under control.

9. Mr. M.L.P., an 81-year-old semi-invalid with "back trouble," was seen because of deafness and tinnitus. He gave a history of having chewed tobacco for almost "all my life." About two years ago he had received a series of penicillin and "mycin" injections for a "cold." For a long period prior to examination he noted a "bad taste" in his mouth. The tongue presented the appearance (see Fig. 13) dorsally of caracul, or black lamb's wool, from the foramen caecum to the tip; the appearance more like that of a two-toned automobile, being black on top and pink along the sides. The pH was 5 and cultures revealed *Candida* sp. but not *Albicans*. He refused biopsy and only reluctantly accepted an alkalinizing regime for his tongue lesion.

DISCUSSION AND ANALYSIS OF 127 CASES OF "COATED TONGUE."

Every one of the cases could give the history at some time or another, varying from four years to the past few weeks, of having had one or several of the antibiotics, including most frequently the broad spectrums prior to examination. Many patients who complained bitterly of a mild sore throat or a constant ache best located in the hypopharynx, came into the office requesting "just a shot of penicillin" to clear up the symptoms. Needless to say, very few even got the antibiotic most sensitive to the actual infecting organism on the tongue, and then only when a serious complication such as submandibular abscess or laryngeal edema ensued.

Though only nine cases could be ascribed to BHT, there were 42 which could be classified as light yellow to brown, and 76 that were white. The BHT people had very few complaints, but when they did, the extensions were more severe than in the other groups. These extensions or concomitant disease manifestations were present in five cases out of the 127. One had an accompanying sub-lingual abscess *not* involving the teeth or sub-maxillary gland; one had acute

edema of the right arytenoid almost completely closing the glottis and responding well to ACTH and Medrol®; a third had bilateral vocal cord edema with hoarseness as a result of the constant clearing of the throat initiated by the presence of the cenosis at the base of the tongue and a feeling of a "lump in the throat"; a fourth (see Case 8) had a severe corneal ulcer, from which *Aerobacter* sensitive only to chloromycetin was isolated, and a uveitis which responded to the usual heroic measures only after his BHT was under control; and the fifth had a mild uveitis responding to typhoid therapy and mydriasis only after his lingual cenosis was cleaned up.

Practically all patients had one or more manifestations of allergy involving the nose, throat or conjunctivae. They are apparently *allergic to their own organisms.* The white tongues most frequently had a yeast, not *Candida,* and many of the yellow-brown ones grew out one or more flavo-organisms, and usually just one significantly resistant organism. Smears were taken in all patients, and not one had Vincent's fuso-spirochetal organisms present. When a purulent sinusitis appeared in several cases, it was treated with aeration and drainage and the appropriate antibiotic, while covering the patient with vitamins and anti-fungal agents. Even then these developed more of coating to the tongue which did not clear up until the antibiotic was discontinued.

In addition to the allergic rhinitis symptoms that practically all patients manifested, other E.N.T. complaints involved external otitis, from which monilia and other resistant organisms were cultured in 15 cases. Referred pain either to the back of the neck, referred to the ear or anteriorly toward the thyroid region, was noted in 34 patients.

Concomitant symptoms in other parts of the body ranged from vaginitis in nine women, pruritus ani and cystitis of equal incidence in each sex; 23 males and 25 females. Other diseases included diabetes in two cases; either uveitis or corneal ulcer, or both, in two cases; duodenal ulcer, submandibular abscess, and laryngeal (arytenoid) edema, glottic trauma resulting in vocal cord edema and hoarseness, one each.

In the 127 cases reviewed there were about 5 per cent

failures in response to treatment. Those cases in which the initial pH was found to be 5, tended to require a longer period for resolution of the lingual cenosis than those in which the pH was 6 or greater. Those that failed to clear up were those who did not conscientiously follow the regime outlined for them, or those who in spite of warnings persisted in obtaining "shots" of antibiotics for minor upper respiratory infections. It is to be emphasized again that persistence both on the part of the physician as well as that of the patient is necessary in order to obtain completely satisfying results.

SUMMARY.

Black hairy tongue is not an isolated disease involving the tongue alone; it is actually a syndrome of the head and neck, with occasional manifestations in the genito-urinary, gastro-intestinal, central nervous system, and with often serious local extensions. The various stages in its development can be unified by the concepts both of *micro-* and *macro-*biocenosis. The heretofore multiple compartments into which the "coated" tongues were divided, frequently caused hair tearing exasperation so that the physician often found himself treating a concept rather than the disease or the patient, can now largely be discarded. Rational treatment and management can be based on physiologic and cenologic principles which greatly simplify the matter. Involved in the syndrome of BHT are:

1. It is practically always initiated by the antibiotics which suppress sensitive micro-organisms, allowing the various resistant bacilli, cocci, and fungi to develop their own microbiocenologic relations in the feathery filimentary papillae of the tongue dorsum. The tip and sides of the tongue are not involved. This parasitism is secondary and not primary; nevertheless, it causes more or less distress to the patient, depending on his resistance and the state of his immunity. The titer of these last two factors determines its manifestations; whether "thrush" in those with weak constitutions, or a "coated" tongue that varies from white, through yellow-brown, to black hairy or "mossy" tongue in those of stronger resistance. The oral acidity probably determined by aciduric

micro-organisms tends to perpetuate this state of cenosis by preventing the normal attrition of the keratinized filiform papillae. The organisms aid in this by forming a mucous blanket, which prevents medication from reaching the source of infection. To maintain the nutritional status of the individual, B complex with vitamin C and sometimes large doses of oral vitamin A are essential.

2. *Allergic reactions* to one's own micro-organisms located on the tongue. These vary from mild nasal stuffiness to the full-blown typical allergic rhinitis, and even purulent sinusitis in patients who have never before experienced "hay fever." The conjunctivae and lids may also be involved, as can the external auditory canals. Frequently there is a moniliasis of the external canal concomitant with the same type of infestation of the tongue. Many patients complain of a "dry" throat, or an irritation of the throat at the level of the cenosis, with a constant desire to clear the throat. Parabromdylamine maleate has been found far superior to any other antihistamine in treating these complaints. The corticosteroids, while effective in controlling the allergic state, also favor spread of fungi.

3. The concept of a *parasitic cenosis* is sustained, but emphasis must be on its secondary nature. A multitude of resistant organisms can be found, both as to variety as well as to quantitative numbers, but more significantly in three cases *Geotrichum* has been isolated. Its clinical similarity to *Candida* is noted, and only by cultural studies can it be differentiated. This is important because treatment of the two is different; iodides for the former, gentian violet for the latter. Out of the series of 127 cases, *Candida albicans* was isolated from the tongue in only two cases.

4. Although all 127 cases studied gave a history of antibiotics at some time during their period of symptoms prior to diagnosis, not one case was found associated with malignancy, and only two had diabetes. One case acquired a duodenal ulcer while awaiting the outcome of his compensation case. One case was presented with associated submandibular abscess by a resistant staphylococcus. An autogenous vaccine was considered, but with rapid recovery of the pa-

tient was not advised. Another case was noted with acute laryngeal edema involving the arytenoid on the right; a yeast, not *Candida*, and *Aerobacter* was isolated from his tongue. One case had bilateral vocal cord edema from constantly clearing her throat. The cenosis at the base of the tongue gave her the sensation of a "lump" or something that would neither go up nor down. Two other cases had an associated eye involvement which did not begin to heal until their tongue lesions had cleared. Most of the individuals noted a fetid or "bad" taste in the mouth. They came in complaining most frequently of a sore throat, and not a few wanted more penicillin. Many of the women complained of aches and sharp pains radiating to the back of the neck and down anteriorly toward the thyroid area. Apparently all of the patients had a lowered resistance to a recent infection, bacterial as well as viral, and most had received one or more "shots" of penicillin. Not a few had pruritus, and some women stated that they had a prolonged course of treatment for vaginitis during the time of their "coated" tongue. The public health aspects of this problem will become more important in the future, as more of our population are exposed to "hidden antibiotics" in our food, and particularly milk products; these factors probably account for many of the cases presented here, and may lead directly to a recurrence of the BHT when the patient is exposed to even minute amounts of penicillin, for example, in milk.

To break the protective blanket laid down by the resistant organisms, mechanical scraping with cup or spoon curette and other novel instruments has been utilized. Emphasis is placed on persistence by both patient and physician. Purified pancreatin in powder form, held on the tongue for periods up to 15 minutes, has been found most useful in preparing lysis of the keratinized filiform papillae. It is much less toxic than podophyllin. Prior to using pancreatin in treatment, the average time for clearing the tongue was about two months. With pancreatin the restoration to normal required a much shorter time, averaging about a week or ten days.

The pH of the oral secretions can easily be determined by

using paper strips specially prepared and indicator chart. In BHT it is found to be markedly acid, up to pH 5, while normal mouths are slightly acid to neutral. During and immediately after using pancreatin powder the reaction is alkaline.

In closing, it would be appropriate to quote some of the conclusions of Makower (p. 782): "The knowledge of biocenology can add new weapons in the fight against infectious disease. In furtherance of this aim it will be necessary, *1.* to influence the humoral and the cellular environment of the parasites inside the animal body (through such factors as pH, hormones, nutritional adjustment, and vaccination); . . . *3.* to influence microbes that may cause disease . . . by appropriate antifungal and microbial therapy (on microbes that have become antibiotic-resistant and have established normal microcenotic relationships); *4.* to influence the establishment of solid immunity in patients treated with antibiotics by simultaneous or subsequent immunization with killed or live vaccines consisting of the appropriate microbes . . ."

Yes, the Greeks had a word for it: *biocenosis*, and the concept will be of increasing importance in the medicine of the future.

Acknowledgments: The opinions expressed in this paper are solely the author's own and he alone is responsible for them; however, I express my thanks to Dr. William R. Bond, of the Medical Department, and Mr. F. Burton Hazlerigs, hospital representative of the A. H. Robins Company, Inc., for expediting the materials, particularly pancreatin, the vitamins and the antihistamine used in this study; also to Miss A. Brim, of the Georgia State Public Health Department, and to Dr. L. Ajello of the C. D. C. in Atlanta, Ga., for help in the cultures, particularly the identifications, and to Dr. John Godwin and Dr. James F. Olley for the pathology descriptions, the latter for taking the photomicrographs in color film. Last, but not least, to Mrs. Nora Brown, of Crawford W. Long Hospital Bacteriology Laboratory, for much of the culture studies.

BIBLIOGRAPHY.

1. WINER, LOUIS H.: Black Hair Tongue. *A.M.A. Arch. of Dermat.*, 77:97-103, Jan., 1958.

2. MAKOWER, H.: Biocenologic Problems in Immunology. *Trans. New York Acad. of Sci.*, 20:765-784, Series II, June, 1958.

3. HEIDINGSFELD, M. L.: Hairy or Black Tongue, Report of 100 Cases. *Jour. A.M.A.*, 55:2117, Dec. 17, 1910.

4. LASKARIS, T., and CURTIS, G. D.: Concerning the Parasitic Etiology of Hairy Tongue. *Jour. Investig. Derm.*, 13:99-107, 1949.

5. PILLSBURY, DONALD M., ET AL.: "Textbook of Dermatology," p. 237. W. B. Saunders Co., Philadelphia, 1956.

6. KLEINERT, MARGARET N.: Hairy or Black Tongue. *Ann. Otol., Rhinol. and Laryngol.*, 55:188-195, 1946.

7. LAYNE, J. A., and WATSON, C. J.: Pellagra in Dogs. *Ann. Int. Med.*, 19:200-205, Aug., 1943.

8. WEIDMAN, F. D.: The Affinity Between Black Tongue and Trichomycosis. *Arch. Dermat. and Syph.*, 18:647-665, Nov., 1928.

9. LASKARIS, T., and CURTIS, G. D.: op. cit., p. 105.

10. SWINBURNE, GEO.: Black Hairy Tongue. *Jour. Laryngol. and Otol.*, 54:386-404, July, 1939.

11. WINER, L. H.: op. cit., p. 97.

12. MONTAGNA, W.: "The Structure and Function of Skin," p. 190. New York Academic Press, Inc., 1956.

13. MAKOWER, H.: op. cit., p. 766.

14. DEBARY, A.: "Die Erscheinung der Symbiose," Tubner, Strassburg, Austria, 1879.

15. ESCHEWEGE, J.: Localization of Generalized Moniliasis in the Brain. *Arch. Neurol. and Psychiat.*, 79:250-263, March, 1958.

16. MAKOWER, H.: op. cit., p. 768.

17. URI, J.: Dermatophytes in Skin and Hairs Produce Antibiotics. *Nature*, 179:1029-1030, London, May 18, 1957.

18. FINLAND, MAXWELL: Current Concepts in Therapy, Antibiotics, III. *New Eng. Jour. Med.*, 258-546, March 13, 1958.

19. ROSENTHAL, A.: Follow-up Study of Fatal Penicillin Reactions. Special Report, *Jour. A.M.A.*, 167:1120, June 28, 1958.

20. YANNOULIS, G. E.: The pH in Otolaryngology, Especially in Papilome of the Larynx. *Abstract Sixth International Congress of Otolaryngology*, p. 408, Washington, D. C., May 5-10, 1957.

21. "Zinnser's T. B. of Bacteriology," p. 849, 11th Ed. Appleton-Century Crofts, Inc., New York, 1957.

22. EDITORIAL: Reactions After Antibiotic Administration. *Jour. A.M.A.*, 166:928, Feb. 22, 1958.

124 West Princeton Ave.

CLINICAL EVALUATION OF PARABROMDYLAMINE MALEATE (DIMETANE®)* IN ALLERGIC AND VASOMOTOR RHINITIS.

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A large portion of the office practice of any otolaryngologist consists of patients with allergic or vasomotor rhinitis. The otolaryngologist and the allergist cannot always differentiate these two varieties of rhinitis. A number of observers have attempted to differentiate the two, and others have put them in one general category. Nasal obstruction of some degree is usually present in all of these patients and frequently associated with one or more related symptoms. The presence of inflammation and hyperemia or pallor, ischemia, and edema of the mucosa of the nasal passages seems to be the common denominator in this large group of patients, whether the cause is allergic, inflammatory, or related to emotional or other causes.

Antihistaminics have been used in the treatment of varieties of rhinitis with good results by a number of observers. This, under some circumstances, has led to the belief that those patients who benefit from the use of antihistaminic drugs have an allergic manifestation. It should be noted, however, that while these drugs may overcome the effects of exogenously administered histamine and apparently counteract the effects of endogenously released histamine-like agents, they have other pharmacological effects. One of these may be more specifically related to the reduction of edema of nasal mucosa.

Halpern[1] has expressed the belief that the primary action of antihistaminics may be that of reducing the permeability

*The parabromdylamine maleate was supplied as Dimetane® by the A. H. Robins Co., Inc., Richmond, Va.

†The opinions or assertions contained herein are those of the writer and are not to be construed as official or reflecting the views of the Navy Department or the Naval Service at large.

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of the capillary wall. He found that an antihistaminic agent prevented albuminuria seen under certain conditions in rabbits—a phenomena thought to be unlikely as a result of histamine. We wonder if the reduction of edema could not be due to the ability of an antihistaminic agent to decrease capillary permeability in the nasal mucosa.

We have observed 171 cases of allergic and/or vasomotor rhinitis in recent months and have followed their response to treatment with the antihistaminic agent, parabromdylamine maleate (Robins[2,3,4,5]). In the classification of response to treatment a definite positive response was considered in those patients who reported their symptoms satisfactorily controlled or asymptomatic as a result of therapy.

MATERIAL.

More than 200 patients were seen in the outpatient clinic of the U. S. Naval Hospital, Newport, R. I., and 171 of these patients were followed for periods up to six or more months. Parabromdylamine was employed in all of them for the relief of their symptoms related to allergic and/or vasomotor rhinitis. The chief complaints of these patients were:

1. Nasal obstruction.
2. Post-nasal drainage.
3. Loss of hearing.
4. Sinus-like pain.
5. Cough.

The group consisted of 112 adults and 59 children, and nasal obstruction was present in each case and frequently associated with other symptoms. Females outnumbered the males 112 to 59, and adults outnumbered the children 112 to 59. Patients were followed at frequent intervals and a six-month or greater follow-up after improvement was made in all of the 171 cases reported.

ASSOCIATED CONDITIONS.

Eustachian salpingitis with definite hearing loss was noted in 62 patients. Treatment in these patients consisted of intranasal Eustachian tube catheterization followed by para-

bromdylamine [Dimetane Extentabs twice a day employed in 50 adults and Dimetane tablets (4 mg.) or Elixir in 10 children]. On this therapy, 46 of the 50 adults improved satisfactorily with but one inflation of the Eustachian tube. It was noted by most of the patients that if the antihistamine was stopped before one week of therapy the symptoms tended to recur. Where this was reported their symptoms were again controlled by the use of parabromdylamine. Eight of the adult women with Eustachian salpingitis were pregnant. All of them reported satisfactory response from their original episode, and six of them had a recurrence later in pregnancy which was again relieved with same treatment.

TABLE I.

171 Patients with Symptoms Related to Allergic or Vasomotor Rhinitis.

Specific Symptoms		Results with Dimetane®	
Eustachian salpingitis	62	Apparently cured (no recurrence in 6 months)	59
Post-nasal drainage	97	Improved	89
Cough	41	Failed to respond	23
Sinus pain	50	Patients preferring Dimetane to other antihistaminics	97
Laryngitis	4	Patients preferring other antihistaminics to Dimetane	5
		Patients who continued to use Dimetane p.r.n.	73
Adult patients	112		
Pediatric patients	59		
Males	59		
Females	112		

Post-nasal drainage and nasal obstruction was noted in 97 patients. Fifty-five of the 62 patients with this complaint had a significant decrease in their symptoms on treatment. Thirty-three of the 35 pediatric cases noted a definite decrease in both the anterior and posterior nasal drainage under treatment. Some patients reported that their nasal secretions became thicker while on the antihistamine, but this was never severe enough to discontinue using the drug.

Cough and nasal obstruction which had not responded to the usual cough remedies was present in 41 patients. Thirty-six of the group improved while taking parabromdylamine but noted a recurrence of cough if the treatment was stopped in

under two weeks' time. Many of these patients continued to use the drug as necessary with gradual complete relief of symptoms in all but one case.

Sinus-like pain with nasal obstruction showed quite rapid response to the drug. Pain was noted in various combinations in maxillary, frontal and ethmoid sinus regions, and for the most part was relieved to a significant degree in 24 hours. Forty-eight cases showed complete relief of symptoms within seven days, but two cases noted no improvement. Several of these cases had actual fluid levels on roentgenogram, but, adequate spontaneous drainage occurred with elimination of mechanical blockage. These sinuses cleared without the use of antibiotics or antral irrigations.

Five patients had marked nasal obstruction due to chemical rhinitis from prolonged indiscriminate use of nose drops. On parabromdylamine, these patients showed improvement within 72 hours of stoppage of local nasal medication, and continued clinical improvement was marked within two weeks' time. All of them had been using nose drops for many months and showed no relief of nasal obstruction with other antihistaminics employed.

COMPARISON WITH OTHER ANTIHISTAMINICS.

Of this group of patients, 97 of the 171 cases found Dimetane® better than any other previously employed antihistaminics. Seventy-three cases continued to use Dimetane® as neccessary with relief of recurrent symptoms. Forty-eight cases had not used other antihistaminics as far as could be determined. Five patients noted better relief with other antihistaminics than with Dimetane.®

SIDE EFFECTS.

Thirteen patients had side effects of significance. Five complained of being drowsy although only one was drowsy to such a degree that treatment had to be stopped. One complained of palpitation and another of severe dryness of the throat. Both of these patients discontinued treatment because of these complaints.

Seven children were treated with Dimetane Elixir over a period of four to six weeks. All noted a marked decrease in symptoms the first week; later, four became refractory to the drug, and symptoms returned while they were still using the agent. This was not reported with the tablets or the Extentabs.

Dimetane tablets, 4 mg. four times a day, did not seem to be as satisfactory as the Dimetane Extentabs, 12 mg. twice a day. The Extentabs usually were effective for 8 to 12 hours. Several patients used an extra 4 mg. tablet to supplement the Dimetane Extentabs medications.

DISCUSSION.

The fact that 87 per cent of this series of patients had beneficial results from the use of the antihistamine parabromdylamine maleate, does not establish the fact that their symptoms were of an allergic nature; nor does it establish the fact that histamine-like substances are responsible for the induration or edema of the mucosa of the nasal passages. It is interesting to speculate upon the concept presented by Halpern that the primary action of antihistaminic agents is in reducing the permeability of the capillary wall. The decrease of local tissue edema of nasal mucosa may be due to this type of action by parabromdylamine maleate.

The findings of Thomas[2] are confirmed in that those patients who had multiple manifestations of allergy showed a higher percentage of response to therapy than those who had a single manifestation. In our experience Dimetane® was of little value in the treatment of the routine common cold.

CONCLUSION.

Eighty-seven per cent of the series of 171 patients with allergic and vasomotor rhinitis and related symptoms were relieved by the antihistaminic, parabromdylamine maleate. Thirteen per cent reported no improvement. Minor side effects occurred in 13 patients or 7.6 per cent. Dimetane Extentabs 12 mg. appeared to be more effective than Dimetane

tablets 4 mg., particularly in that the Extentabs had a duration of action for 8 to 12 hours.

REFERENCES.

1. Halpern, B. B.: *Acta Allergol.*, 1:3, 1948; Hamburger, J.; Halpern, B. N., and Neal, J.: *Compt. Rend. Soc. De Biol.*, 142.

2. Thomas, J. W.: Para-Bromdylamine Maleate (Dimetane®). A Clinical Evaluation: Report of 140 Cases. *Ann. of Allergy*, 16:128-134, March-April, 1958.

3. Dann, Sidney; Bromn, R. R., and Ruchocki, A. D.: Clinical Experience with Parabromdylamine Maleate (Dimetane), a New Antihistaminic. *Jour. of Allergy*, 29:511-523, Nov.-Dec., 1958.

4. Horstman, H. A.: Clinical Observations with Dimetane. *Amer. Pract. and Digest of Treat.*, 10:96-97, Jan., 1959.

5. Kreindler, Louis; Ghory, J. E., and Bernstein, I. L.: Treatment of Allergic Disorders with a New Antihistamine: Parabromdylamine. *Antibiotic Med. and Clin. Therapy*, 6:28-31, January, 1959.

AMERICAN ASSOCIATION FOR CLEFT PALATE REHABILITATION.

The American Association for Cleft Palate Rehabilitation will hold its Eighteenth Annual Convention at the Brown Palace Hotel, Denver, Colo., Thursday, Friday and Saturday, May 12, 13 and 14, 1960.

This Association is composed of medical, dental and paramedical specialists who are interested in the rehabilitation of persons with cleft lips and palates.

ANTIHISTAMINIC THERAPY.

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HISTAMINE.

It is generally believed that histamine is produced or released in the tissues as the result of allergic reactions, trauma, infection, fever, stress, physical agents and emotions.[1] Patients with allergic manifestations appear to have a diminished tolerance to histamine. It is known, for example, that in such patients small amounts of histamine will precipitate an attack of bronchial asthma. Dragstedt and Mead[2] demonstrated an increase in the histamine level of blood and lymph of dogs after anaphylactic shock. The amount demonstrated was sufficient to account for the reaction. Similar studies have been reported by Code[3] who used guinea pigs rather than dogs.

HISTORY OF ANTIHISTAMINES.

Because of the important relationship of histamine to the allergic reaction, many attempts have been made to counteract or to antagonize the effects of histamine in the body. The first successful use of an antihistamine was reported by Fourneau and Bovet[4] in 1933; however, the phenolic ethers with which they were working proved to be too toxic for general use. Later, compounds containing the ethylenediamine radical were developed which were satisfactory. Quite a variety of antihistamines are now available; most of them belong to one of the three chemical groups.[5]

CHEMISTRY.

The three groups are ethylenediamine derivatives, ethanolamine derivatives, and monamines. Examples, identified by trademark, are the following:

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Ethylenediamine derivatives: Antergan, Chlorothen, Diatrin, Histadyl, Neo-Antergan, Neohetramine, Phenergan, Pyribenzamine, Pyrrolazote, Tagathen, Thenfadil, Thenylene.

Ethanolamine derivatives: Benadryl and Decapryn.

Monamines: Chlor-Trimeton and Trimeton.

In addition, there are a few other antihistaminic agents which are unrelated chemically to the three classical groups. Examples of these miscellaneous compounds are Antistine, Chlorcyclizine, Di-Paralene, Perazil and Thephorin.

RESPIRATORY ALLERGY.

The most satisfactory response to the antihistamines has been noted in patients with seasonal hay fever. Better results have been observed when pollen of trees and grasses rather than of ragweed is the causative factor, because of the lesser degree of exposure. Response, therefore, is more or less proportional to the degree of exposure. Secondary factors, especially other sensitivities, may play a great part in influencing the effectiveness of the drug.

The response in chronic perennial nasal allergy has not been as encouraging as in hay fever. The rhinorrhea is usually most modified. In some cases, the dryness of the nose may be objectionable.

FREQUENCY OF ADMINISTRATION AND SELECTION OF DRUG.

The average duration of effectiveness of most antihistamines is about four hours. Because of drowsiness as a side effect of some of the more potent agents, ambulatory patients in the past have usually been treated with repeated doses of drugs in the ethylenediamine group or with small doses of one of the ethanolamine derivatives. The recent development of a triple antihistamine, containing one representative compound from each of the three groups in a special base from which a constant amount of medication is liberated over a period of eight hours, may simplify not only the dosage

schedule but also the selection of the agent. The new preparation is known by the trademark, "Tridecamine Dospan."*

HUMAN BIO-ASSAY.

After determining that the antihistaminic agents are liberated from the base over an eight-hour period of time, Jones and his associates[6] measured the size of the wheal produced by 0.1 cc. of a 1:10,000 concentration of histamine diphosphate in aqueous solution, injected intradermally in the forearm immediately before administration of Tridecamine Dospan and repeated every two hours for a period of 14 hours. During the entire period of observation, the wheals were always smaller than prior to treatment in the group receiving Tridecamine Dospan. Untreated subjects were used as controls. In this group, the wheals not only were not smaller, they actually became larger as the series of histamine injections was repeated.

CLINICAL EXPERIENCE WITH TRIDECAMINE DOSPAN.

Preliminary trials with Tridecamine Dospan were begun in the hope that the compound would produce prolonged symptomatic relief in those patients capable of responding to antihistaminic therapy, and that side effects would be virtually eliminated. The hope for relative freedom from side effects was based upon two theories; the first was that very few people would be sensitive to agents from more than one of the three major chemical groups. Accordingly, the side effects should not be additive, although the antihistaminic or therapeutic effects would be. Secondly, the gradual release of a medication to which a patient is sensitive would be less likely to produce severe side effects than would the rapid release of the same medication. Indeed, this phenomenon is best illustrated in the case of nicotinic acid, which can be released from the same base, usually without producing flushing.[7]

*Tridecamine is the trademark of The Wm. S. Merrell Company, Cincinnati 15, O., for a combination tablet containing 20 mg. each of doxylamine succinate, pheniramine maleate, and pyrilamine maleate; Dospan is the trademark of The Wm. S. Merrell Company for the special sustained-release base.

The clinical response to Tridecamine Dospan has been highly gratifying. The patients were all suffering from nasal allergy. There were 92 males and 133 females, all adult. The dosage varied from one tablet every eight hours to one tablet every 12 hours. In most cases, only two tablets per day were required. The duration of therapy varied from ten to 30 days. In this group of 225 patients, there were only 15 (6.6 per cent) therapeutic failures. In 30 other patients (13.2 per cent) there was only moderate symptomatic improvement. In the remaining 180 patients (80.2 per cent) the symptomatic relief was excellent.

Perhaps the most important reason for popularity of the new preparation among the patients was the dependable relief of nocturnal nasal symptoms. Drowsiness was extremely uncommon and when it did occur, did not interfere with ordinary activity.

SUMMARY AND CONCLUSIONS.

Tridecamine Dospan is a new triple antihistamine from which the medication is released gradually over a period of eight hours to produce an antihistaminic effect for 12 hours or more. In a series of 225 patients suffering from nasal allergy, it produced highly gratifying symptomatic relief in 80.2 per cent, moderate relief in another 13.2 per cent, and no relief in only 6.6 per cent. The dose is one tablet every eight to twelve hours.

REFERENCES.

1. HANSEL, F. K.: "Clinical Allergy," pp. 38-41. The C. V. Mosby Company, St. Louis, 1953.

2. DRAGSTEDT, C. A., and MEAD, F. B.: The Role of Histamine in Canine Anaphylactic Shock. *Jour. Pharmacol. and Exper. Therap.*, 57:419-426, 1936.

3. CODE, C. F.: The Mechanism of Anaphylactic Allergic Reactions. *Ann. Allergy*, 2:457-471, 1944.

4. FOURNEAU, E., and BOVET, D.: Recherches sur l'Action Sympathicolytique d'un Nouveau Derive du Dioxane. *Arch. Internat. de Pharmacodyn. et de Therap.*, 46:178-191, 1933; abst., *Compt. Rend Soc. de Biol.*, 113:388, 1933.

5. HANSEL, F. K.: "Clinical Allergy," pp. 707-721. The C. V. Mosby Company, St. Louis, 1953.

6. JONES, T. L.; DALE, L. B., and CHRISTENSON, G. L.: Duration of Effect of a New Sustained Release Antihistamine, Tridecamine. To be published.

7. O'REILLY, P. O.; CALLBECK, M. J., and HOFFER, A.: Sustained-release Nicotinic Acid (Nicospan). Effect on (1) Cholesterol Levels and (2) Leucocytes. *Canadian Med. Assoc. Jour.*, 80:359-362, March 1, 1959.

GILL MEMORIAL EYE, EAR AND THROAT HOSPITAL.

The Gill Memorial Eye, Ear and Throat Hospital has just completed its Thirty-second Annual Spring Congress in Ophthalmology, Otolaryngology and allied specialties. There were forty-two states, England, Canada and several foreign countries represented. There were twenty-one guest speakers, sixty lectures and closed circuit televised surgery during the five and one-half days of the Congress. In 1960, the Thirty-third Annual Spring Congress will be held from April 4 through April 9.

ABSTRACT OF THE PROCEEDINGS OF THE EIGHTIETH ANNUAL MEETING OF THE AMERICAN LARYNGOLOGICAL ASSOCIATION.

Hot Springs, Va., March 8-9, 1959.

FRED W. DIXON, M.D.,
President.

SAMUEL SALINGER, M.D.,
Abstract Editor.

OSTEOMYELITIS OF THE FRONTAL BONE.

HARRY P. SCHENCK, M.D.

The impact of the antibiotics on the incidence, course and therapy of osteomyelitis of the frontal bone was described by Dr. Schenck in an analysis of 56 cases divided into three decades: 1929 to 1938, 1939 to 1948, and 1949 to 1958. His tables showed a decline in the number of acute fulminating osteomyelitis cases from nine in the first period to none in the third period. The offending organism in all types of cases in all three periods was most often the staphylococcus aureus with hemolytic streptococcus in second place.

Emphasis is placed on adequate X-ray studies which require expert interpretation because the antibiotics result in bony changes that differ from those seen in the pre-antibiotic days. These changes may be detected within five days of the onset, but the typical moth-eaten appearance of the bone, previously a constant finding, is now replaced by signs of deossification, decalcification and blurring of the outlines of the sinus.

Sensitivity tests are mandatory in selection of proper antibiotics.

Dr. Schenck attributed some of the cases to injudicious probing of the sinus in the acute stage.

As for therapy, he has found that simple drainage with primary removal of diseased tissue, and minimal removal of bone, was sufficient, as long as drainage into the nose is effected via enlargement of the duct and clearing of adjacent ethmoid cells. In most cases sequestrums were permitted to remain *in situ* since subsequent X-rays showed restoration of the bone by reason of invasion of new vessels.

X-ray pictures, taken at monthly intervals up to one year, reveal gradual disappearance of the translucent zones with replacement by new bone.

DISCUSSION.

Dr. Oliver Van Alyea, while agreeing that the antibiotics have proven tremendously effective in limiting both the incidence and the gravity of osteomyelitis, nevertheless sounded a note of warning against complacency. Occasionally an intracranial complication will develop in cases thought to be under control. The emphasis placed on adequate drainage is fundamental, but Van Alyea felt that external drainage could well be supplemented by intranasal irrigation via the frontal sinus cannula. Some cases are due to inadequate or incompetent initial surgery on the sinuses, as evidenced in several cases cited by the speaker.

Dr. Henry M. Goodyear questioned Dr. Schenck about the length of time the tube was left in place. He thought it was stated to be as much as six months, whereas Dr. Schenck stated it was six weeks. Dr. Goodyear feels that sufficient drainage can be obtained by removing the anterior ethmoid cells, avoiding traumatizing the nasofrontal duct and thus dispensing with the tube. He has found that such a passageway can be maintained for many years. He has employed the intransal removal of these cells even in acute cases, and has never had an intracranial complication result. He asked Dr. Schenck whether external drainage via a tube could not be dispensed with if adequate drainage has been established intranasally.

Dr. W. Likely Simpson also has employed intranasal drainage even in acute cases, introducing a tube into the frontal sinus which is left in place for several days. Dr. Simpson then showed a number of slides of cases of osteomyelitis and several different surgical procedures for their management.

Dr. Daniel C. Baker, Jr., stated that his experience has been similar to that of the essayist, confirming particularly the value of early external drainage of acute cases. He personally has not seen a case of fulminating osteomyelitis in ten years. He also has been using a tube for drainage via a large opening into the middle meatus and avoiding the nasofrontal duct. A polyethylene tube is well tolerated for the six weeks period it is in place.

Dr. Schenck, in closing the discussion, stated that while he doesn't doubt Dr. Van Alyea's skill in passing a frontal sinus catheter, he does doubt that many others possess the same degree of skill. His experience

has been that many cases of osteomyelitis have developed following this procedure. The use of an indwelling tube is certainly not original with himself, having been employed by others for many years. Dr. Goodyear's point with regard to removal of anterior ethmoid cells is well taken, and this usually occurs when one is fashioning the drainage channel from above with the rasp. Maintenance of the duct and its bony walls is also an extremely important item. His objection to going in from below via the ethmoid cells is that one fails to see the interior of the frontal sinus and to deal adequately with its pathology. This is particularly important when there is a defect in the posterior wall. As for drainage via the anterior wall, this is urgent in acute cases when there is pus under pressure in the sinus. As for the much feared fulminating cases, which are fortunately rare, there is little that can be done aside from the external trephine and antibiotics. These cases in the past have been rapidly fatal.

AN EXTRA LARYNGEAL APPROACH FOR CERTAIN BENIGN LESIONS OF THE LARYNX.

LEROY A. SCHALL, M.D.

An extra laryngeal approach for the removal of certain benign lesions of the larynx was demonstrated by Dr. Schall. It was successfully employed in four cases: a neurofibroma, a chemodectoma, an internal laryngocele and an external laryngocele.

The procedure consists of an exposure of the thyroid ala via a horizontal incision, elevation of the internal perichondrium from the ala, removal of the cartilage from the midline to the oblique line, and from the superior margin down to include at least two-thirds of the cartilage and an incision through the thyro-hyoid membrane, which permits blunt dissection of the growth of a cyst. If it is a laryngocele, after exposure of the fundus, it may be opened, a finger inserted into its lumen and removal accomplished by both sight and feel. A cyst may be aspirated and then removed the same as a laryngocele.

Tracheotomy may be indicated preoperatively if there is some dyspnea; otherwise, it is performed at the conclusion of the operation.

The advantages of this operation are adequate exposure, no intralaryngeal trauma and no intralaryngeal complications.

DISCUSSION.

Dr. John Conley commented on the advantages of this technique, which is similar to a horizontal pharyngotomy and has the advantage of avoiding direct trauma to the intralaryngeal structures. The management of a large cyst and the laryngocele by this procedure seems more than adequate with the help of a finger introduced into the cavity as described. Dr. Conley believed that the indications for this operation depend largely upon the accuracy of the diagnosis. Often the question arises as to whether one was dealing with a cyst, a tumor, or a laryngocele. This might prove embarrassing if the mass happened to be malignant. He has seen cases of the "iceberg" type where the growth was entirely submucosal, and another in which a cancer developed in a laryngocele. The technique described seems quite simple. The incision assures security so far as the superior laryngeal artery and nerve are concerned, the latter being important to conserve. Removal of an adequate amount of cartilage affords sufficient working space. Naturally, great care must be taken to avoid injury to the intralaryngeal structures. He asked Dr. Schall whether it might not be a good idea, instead of removing two-thirds of the alar cartilage, to come to the midline and swing the alar cartilage back on a muscle pedicle (the inferior constrictor) thus opening up the entire extralaryngeal space to work in.

Dr. Schall stated that he had borrowed the procedure from the transhyoid or intra-hyoid pharyngotomy approach. He agreed that there is some risk in the extra laryngeal approach insofar as the possibility of encountering a malignancy is concerned. In his two tumor cases, one of 20 years' standing, the other of ten years, he felt that preliminary biopsy was not urgent in view of their chronicity. As for differentiating between a cyst, laryngocele or tumor by means of X-rays, his radiologist, Dr. McMillan, has been unable to determine, since they all cast approximately the same shadow. He, too, has seen cases of carcinoma develop in a laryngocele which was not recognized until after the extirpated mass had been removed. Dr. Schall has known of Dr. Orton's procedure, as well as that of Dr. O'Keefe, in which the thyroid cartilage is split. He missed seeing Dr. Cardwell's movie on laryngocele at the San Francisco meeting and is, therefore, unable to comment on it; however, he is satisfied that the procedure he has just described was adequate and successful for the types of cases in which they were employed.

CARCINOMA OF THE NASOPHARYNX.

DR. L. Q. PANG (By Invitation).

In presenting an analysis of 34 cases of carcinoma of the nasopharynx, Dr. Pang brought out an interesting item relating to the high incidence in the Chinese who constituted 80 per cent of his series. Similar findings were noted in reports from centers as far apart as New York and Formosa.

The most common symptoms noted were enlarged cervical nodes, unilateral nasal obstruction, and ear symptoms. Diagnosis is frequently missed because the lesion is not sought for,

and frequently excapes notice despite nasopharyngeal examination. Biopsies are often inadequate and have to be repeated until the true nature of the lesion is disclosed. Pang has found the average time from onset of symptoms to positive diagnosis averaged six-and-one-half months.

The lesions which are mostly transitional cell carcinomas are quite radiosensitive, yet their curability leaves much to be desired. The author favors the transpalatal approach for visualization of the area and the direct application of surgical diathermy. His results have not been too favorable, since remissions after primary abatement have been very frequent. Only six cases survived five years, two of these having already had a remission, leaving only four cases reaching a six year survival without symptoms (12 per cent). As for neck dissection, Pang feels that it is indicated only in cases where the primary lesion is definitely controlled by irradiation.

DISCUSSION.

Dr. Daniel S. Cunning commented on the fact that the diagnosis and therapy of this condition have changed very little in the past 25 or 30 years. We are still baffled by the delays encountered in establishing a diagnosis, a fact which is borne out by Dr. Pang's statement which revealed an average lapse of six months from the onset of symptoms. While it is true that in the beginning the patient himself is at fault in failing to seek advice when the early symptoms manifest themselves, in the main, the chief responsibility lies with the physician who first sees the patient, and the laryngologist whose examination of the nasopharynx is perfunctory. An isolated cervical node or unilateral ear symptoms should at once alert the surgeon and should always arouse suspicion of a primary lesion in the nasopharynx. While the percentage of five year cures is pathetically low, experience has shown that the cases diagnosed early have at least a 50-50 chance for recovery. The high incidence in Orientals is more than an incident. More research along anthropological, dietary and hormonal lines is necessary to explain adequately this unusual predisposition. The treatment of cervical metastases depends upon the type of growth, the more radio sensitive tumors being amenable to irradiation, whereas the more adult types require radical neck dissection. The palatine approach described by Dr. Pang is very useful in the visualization of the area but needs evaluation through the observations of many surgeons in this field. Other points that can bear stressing are the importance of repeated biopsies if the initial effort is fruitless, and the need for disregarding psychoneurosis as a diagnosis for symptoms not yet fully explained in the beginnig.

Dr. Daniel C. Baker asked for more information about the patient who suffered a radio necrosis of the brain. During the past year he himself had had a patient with a lymphosarcoma of the nasopharynx who had been treated with cobalt and had obtained a good primary result; however, about three months later, he collapsed on the golf course with a sudden attack of vertigo, lapsed into coma and expired within 24 hours. Autopsy revealed a radio necrosis of the brain. The case was the third of its kind reported at the New York Neurological Institute.

Dr. Samuel Salinger was impressed by the high incidence of this condition among the Chinese, which is apparently characteristic of the race, since similar reports have emanated from as widely separated locations as New York and Formosa. Not much was said about X-ray diagnosis. In his experience roentgenography has not been very helpful, except where the case had advanced to the stage where bony necrosis can be visualized. Possibly laminography may be of some help in differentiating degrees of thickening of the soft tissues. Experience has shown that early negative biopsies cannot be relied upon since in many cases the growth is subepithelial, and it may require several attempts with deeper bites to reach the tumor tissue.

Dr. Julius W. McCall inquired as to whether there was a connection between this condition and the chewing of betel nuts.

Dr. Louis H. Clerf, being interested in cytological diagnosis, suggested placing the patient in the posture as for a Proetz treatment, instilling a saline solution and then drawing it off for a Papanicolaou stain.

Dr. Eugene S. Hoff stated that he and the late Dr. Morrison had taken a number of smears from the nasopharynx for Papanicolaou staining and had obtained positive diagnosis in some cases. In line with what Dr. Salinger said, in the San Francisco area Dr. Hoff had noted an incidence of five times among the Chinese as compared with the Caucasian.

Dr. Robert E. Priest, whose community is predominantly Nordic, found the Chinese component practically negligible. The incidence among the Nordics was found to be only one in 386,000 admissions. The one case he saw was treated by implantation of radon seeds with a total dosage of 4000 *r*. Subsequently an additional 3000 *r* was given by the same method, and the patient is alive and well after ten years. A review of the biopsies confirmed the original diagnosis of the malignancy.

Dr. Pang explained that the fenestration of the palate as an approach to the involved area was very satisfactory. It is particularly so for cases that come from a distance and must be followed up by their local doctor. The fenestra makes it possible for the general practitioner, or even the patient himself, to see the operated area. The one case of radionecrosis of the brain had received deep X-ray thrapy to the cervical nodes, followed later by treatment with the 1,000,000-volt machine at the University of California. His experience with cobalt therapy, which is limited, makes him feel that it will supplant deep X-rays. As far as betel nut chewing is concerned, none of his patients had the habit to his knowledge. He has tried cytologic examination of smears, and feels that it has great promise. As for the radon seed therapy, Dr. Pang feels that with the palate fenestra it is possible to apply more accurately any therapy you choose, and certainly radon seeds can be implanted much more accurately by this method.

THE SIMULATED LARYNX.

C. J. CAMPBELL, M.D.,
and
JOHN A. MURTAGH, M.D.

Dr. Campbell and Dr. Murtagh reported the results of their investigations with the Gooch tube in the production of simu-

lated vocal cord action. A large tubing was used in order to keep the frequencies low and the action of the stretched lip observed with a high speed camera in various phases with the air flow recorded at both input and output. Changes in the dimensions and contours of the inferior surface of the glottis were observed with their effects on frequency and wave form, which pointed to the action of the subglottic portion of the thyro-arytenoideus muscle suggesting its determinant of the frequency of vocal cord vibrations.

DISCUSSION.

Dr. Gordon D. Hoople pointed out several aspects of the experiments which might be better appreciated by the viewers of the movie if samples of the sounds of the reeds could have been included on the sound strip. He also suggested that the author's summary might well include a brief description of their experimental design. A change in the title was also suggested, which would more accurately describe the work done. Since further experiments have been projected by the essayists, no doubt the improved photographic equipment they have in mind will add much to the value of their studies.

Dr. Murtagh explained that they have recently been photographing the subglottic region in animals with the same high speed action analysis camera at 8000 pictures per second, which produced very instructive and revealing data. The results differed from the previous experiments, which were taken at 2000, because the input pressure in the latter cases was at low frequency and thus did not require any higher speed photography. Since the studies are continuing, they have tried to start from the bottom, hoping by studies at various speeds, frequencies and tensions to stumble on something of practical value.

SURGERY OF THE NECK FOLLOWING RADIATION THERAPY FOR CANCER.

WALTER P. WORK, M.D.

Dr. Work reported his results with surgery of the neck in cancer cases previously unsuccessfully irradiated. These cases were divided into three groups, namely nonarrested or recurrent cases of laryngeal cancer, early or late, cancer controlled by irradiation but followed by complications, and pharyngeal cancers partially or fully arrested but presenting definite indications for surgery.

Surgery in cases operated upon less than one year following

irradiation presented fewer difficulties than those which were operated at later periods. In the latter, the dissection was more difficult and the frequent postoperative pharyngostomas necessitating plastic closures by flaps, etc., attested to the devitalizing effects of the irradiation.

The indications for surgery in the arrested cases were general debility, dysphagia, edema and induration and frequently evidences of thyroid necrosis. Healing in these cases was delayed and fistulas frequent.

Of the 22 patients comprising all three groups, 15 are living and well for periods of five months to nine years. Three cases died of other causes one to three years following surgery, while the remaining four died of cancer.

It was noted that cases operated soon after irradiation continued to manifest the late changes due to the irradiation, such as atrophy of the skin and the appearance of telangiectases.

DISCUSSION.

Dr. Stanton A. Friedberg demonstrated the effects of irradiation with a series of slides showing particularly the endothelial proliferation in the blood vessels with eventual obliteration and the fibrosis of the supporting tissue. Since these changes are slow in developing, it probably accounts for the comparative ease in dissection of the cases operated early and the complications following surgery at a later date. Dr. Work's results justify the rationale of undertaking surgery in all cases where prior irradiation has been unsuccessful, even when attended by local complications.

Dr. LeRoy A. Schall stated that his experience with post irradiation surgery has been contrary to that of the essayists; in fact, in many cases it was disastrous. He showed a number of slides depicting the bad results in several cases that had been previously irradiated, the surgical wounds failing to heal after break down of the tissues. While it is true that in a few cases one may get by, it is still a matter not to be taken lightly, because the breaking down of the operative field with all that it entails leads to demoralization of the patient, dissipation of life savings and a period of frightful suffering before the end.

Dr. Paul H. Holinger pointed out the importance of visualizing the possible reparative and reconstructive measures that may have to be employed postoperatively. It might be wise, therefore, to anticipate such measures and to do some preliminary reconstruction, such as raising and resuturing adjacent skin areas which might be needed for adequate closure following resection.

Dr. Julius McCall called attention to a serious complication that may ensue in these cases, namely, a spontaneous rupture of the carotid artery. In one year's time four such cases were seen at his hospital. Prompt action on the part of the staff controlled them and none of them de-

veloped cerebral complications after ligation of the common carotid; however, two of the patients died of the cancer, and the other two have shown no extension of the process in the past year and a half. Dr. McCall showed pictures of several cases where post irradiation neck surgery resulted in sloughing and poor healing. He also suggested the preliminary raising of a skin flap for subsequent use in covering the wound.

Dr. John J. Conley stated that the issue is critical, since a number of patients and physicians will resort to primary irradiation, and some cases have been known to respond. The failures, however, are dumped in the lap of the surgeons who have to cope with a situation that is more grave than the original. He also agreed that preliminary plastic procedures are of great value. One must anticipate the possibility of poor healing in an irradiated area. Knowing exactly how the irradiation was given, the dosage, portals, filters, etc., may be helpful in this connection. Then, too, an appraisal of the condition of the neck tissues may be of help—trouble may be expected if they appear hard, board like, avascular and fixed; also the condition of the mucous surfaces must be evaluated. The extent of the proposed surgery will have a bearing on the degree of anticipated trauma, the more extensive the resection the greater the trauma and the more the need for vital tissues as covering. Dr. Conley described several types of flaps that may be employed in these cases. Involvement of the carotid arteries may be prevented if they are adequately covered by healthy vital tissues, affording a good vascular bed.

Dr. Samuel Salinger felt that the discussion might create the impression that irradiation for carcinoma of the larynx is never indicated. This is not the case. Even Dr. Schall has reported a series of localized cord cancers which were cured by irradiation. Cases in which the lesion is limited to one cord, is superficial, non-invasive with the mobility of the cord unimpaired, are good prospects for cure through irradiation. The evil results following irradiation are in many cases due to improper techniques. There are some radiologists who believe that there is a certain stage in the irradiation therapy at which one can determine whether or not the lesion is responding and which is a safe time to stop and resort to surgery. Just how this point is arrived at seems more or less vague. Were it more definite the prospects for successful surgery would be improved. Dr. Work's report shows, at least, that considerable progress has been made in irradiation techniques. The results in former years were uniformly bad.

Dr. Work replied to Dr. Friedberg's query as to exploratory surgery in these cases. He did not think that an irradiated larynx should be opened because of the risk of infection. It is better to do the biopsies by direct laryngoscopy. Preliminary reconstructive measures, as mentioned, would certainly lessen postoperative complications. As for hemorrhage, he had seen only one such case where the lingual artery ruptured but was promptly taken care of. As for irradiation in early cases, as mentioned by Dr. Salinger, he had to admit that the group of cases he has presented were not a truly representative group, being more selective. He was quite sure the roentgenologists would give him a battle were they in the audience at the time. He certainly would admit that many cancer cases have yielded most satisfactorily to irradiation therapy.

NASAL INFECTIONS IN PATIENTS WITH ASTHMA AND ASTHMATIC BRONCHITIS.

K. M. SIMONTON, M.D.

Dr. Simonton studied the relationship between infection of the nasal sinuses and asthma in two groups of patients seen at the Mayo Clinic. The first group of 252 patients yielded 186 who were referred for a rhinologic examination and among whom there were infections of the sinuses in 24, hay fever, vasomotor rhinitis or polyposis in 113 and significant anatomic abnormalities in 19. Nasal operations were done on 18 of this group, and advised in three additional cases. The remainder were treated non-surgically.

The second group consisted of 128 patients with a diagnosis of asthma or asthmatic bronchitis, on whom some rhinologic procedure had been performed. Information on the results was available in 109. In addition to the treatment of the nose and sinuses, the usual therapy for asthma was administered.

The results indicated that when infection or other nasal abnormalities were properly corrected as indicated, an improvement in the asthmatic status was noted in 44.6 per cent. The results were better in females than males; also it was observed that the highest incidence of favorable results was found in the age group below 20 and above 60. Simonton explains this paradox in this manner: In the young, the nasal disease has not developed to the degree warranting surgery, whereas in the old, reduced exposure to irritants incident to occupation may have been the factor.

DISCUSSION.

Dr. Francis W. Davison pointed out the complexity of the topic which makes it somewhat controversial. There has always been a conflict between the allergist and the rhinologist as to relative value of etiologic factors and the methods of employing antigens for desensitization.

The indications for surgery are indeterminate and variable in different hands—yet, surgery is often inevitable when other therapy has failed. Dr. Davison has usually given antibiotic and steroid therapy a trial before resorting to surgery. He would like to have Dr. Simonton's views in this connection. It was stated that 50 per cent of the asthmatics obtained relief from surgery. Dr. Davison is concerned about the other 50 per cent. Why did surgery fail? It was interesting to note that females

responded better than males. This had not been the case in his own experience.

Dr. Francis L. Weille called attention to a report he submitted several years ago wherein improvement in the asthma following surgery was noted in 52 per cent for six months up to 20 years. The longer the patients were followed the higher the percentage of improved cases. The overall results in the nose were 75 per cent. He inquired of Dr. Simonton just how other therapy was applied and why he thought that nasal surgery was the maximum therapy just because a high percentage of the asthmatics had sinus disease. What happens to those who get better without nasal surgery of any kind? He also would like to know whether the ethmoid operations were all intranasal or external, and whether the middle turbinate was removed.

Dr. Oliver E. Van Alyea recalled a study made at his hospital some years ago in which all asthmatics were subjected to sinus exenteration whether or not they appeared to be involved, on the basis of a direct tie-up connection between the two. This project failed, and the results were never published. Many of these patients admitted having temporary relief, but all of them soon lapsed back to their asthmatic states. Some of them were later treated for sinusitis, probably resulting from the surgery. At the present time they have been doing a number of Caldwell-Luc operations for the removal of polyps and diseased tissue from the antrum in asthmatics. If no polyps are found the ethmoids are not touched. On the subject of nasal polyps there has been some disagreement between the allergists and the rhinologists, the former being in the main, opposed to the procedure.

Dr. Simonton replied to Dr. Davison's question relating to the use of antibiotics and steroid therapy by stating that their patients had all been through a very comprehensive program of medical therapy before surgery was ever considered. When indicated, antibiotics were employed, and in a group of 252 patients there were only 18 on whom surgery was performed. This will perhaps answer Dr. Weille's question as to the point of maximum therapy. Some cases showing evidence of sinus disease were not subjected to surgery when other therapeutic measures were sufficient to afford them relief. Surgery was indicated only when a chronic suppurative process was established. Therapy for allergy was carried out through desensitization and the avoidance of irritants. Polypi were present in most of the cases that were operated. Dr. Simonton feels that patients who have had polypi for a long time almost invariably develop an infection. Surgery primarily was intranasal ethmoidectomy, preserving the middle turbinate and more recently transantral ethmoidectomy with attention to the antrum pathology.

THE DIAGNOSIS OF CHRONIC INFLAMMATORY LESIONS OF THE SPHENOID SINUS.

J. H. MAXWELL, M.D.
and
B. JAY HILL, M.D.

Dr. Maxwell and Dr. Hill presented an analysis of 16 cases of involvement of the sphenoid sinuses due to chronic infec-

tion, mucocele, malignant growth of the nasopharynx and tumors of the pituitary gland. Several aspects of the problem stand out in sharp relief, namely, the prominence of headaches, usually fronto-occipital, of long duration; the failure in most cases to suspect the sphenoid and the delay in having a thorough rhinologic examination and the value of good roentgenologic studies. These points were well brought out by the essayists in the detailed documentation of their cases. The headaches which they described were most severe during the night, and frequently associated with pain in the eye. Ocular disturbances such as paralysis of the extrinsic muscles, optosis and proptosis were prominent symptoms in cases of expanding tumors.

X-rays frequently demonstrated osteitis and osteomyelitis of the sphenoid walls. In all cases a thorough examination of the nasopharynx yielded valuable information, and when intranasal surgery failed to accomplish the eradication of the disease an external transethmoid approach yielded the desired results.

The authors stressed the importance of having an awareness of the possibility of sphenoid sinus involvement at all times and the value of a thorough and complete examination.

DISCUSSION.

Dr. Arthur W. Proetz approved the essayist's searching analysis of a restricted number of varying types of cases. His own reaction was to point out some salient facts pertinent to the subject. *1.* A sphenoid infection is like a fire in a telephone exchange. The symptoms show up in the outlying districts and are apt to be referable to the regions supplied by the important nerves and vessels related to the sinus. *2.* Low grade infections may go on for years undetected because of the location of the sinus and nature of its tissues. *3.* A thick front wall does not necessarily imply thickening of the remaining walls which in many instances are very thin. *4.* Radio opaques may yield significant information by demonstrating fitting defects. *5.* Laminagrams have proved disappointing because of the extreme variability in the thickness slope and distribution of the bony walls. *6.* The value of good X-ray pictures cannot be overemphasized, especially when analyzed by an understanding radiologist. 7. Fungus infection, mentioned by Dr. Maxwell, is more common than generally believed, has a predilection for the posterior sinuses and characterizes the mild chronic types often with allergic manifestations. Looking at the sphenoid and its vital surroundings, Dr. Proetz commented that we were lucky when nature put a couple of nice rugged antrums out in front to divert the passing microbe and the adventuring surgeon from the sphenoid.

Dr. W. Likely Simpson has found the use of contrast media of great

value, and prefers to instill it into the antrum via the natural ostium. As for surgery on the sphenoids, he believes the best results can be obtained only by removing the floor and intersinus septum, as well as the anterior wall.

Dr. John E. Bordley reported that in his service most of the sphenoid sinus cases have been referred from the Neurological Service, which indicates the direct connection between infection of the sphenoid and the intracranial neurological network. Recently they have had two mucoceles of the sphenoid with extension into the ethmoids. Dr. Bordley has found the nasopharyngoscope to be of great value in studying the postnasal and sphenoid area.

Dr. Edwin N. Broyles recalled a case reported in Baltimore some years ago, in which a patient developed diabetes from which he succumbed, and at autopsy the sphenoids were found to be filled with purulent material. It was thought that possibly the irritation of the pituitary had something to do with the development of the diabetes. He asked Dr. Maxwell whether the diabetes in the second case he reported had cleared up after removing the mucocele.

Dr. Maxwell was impressed by Dr. Proetz's reference to the frequent finding of a thick anterior wall and a thin posterior wall, which has often been noted in a similar way with the mastoid. He wondered why the tendency to eburnation of the outer wall should be associated with pressure atrophy and often osteomyelitis of the inner wall. He does not employ contrast media routinely, although conceded their value when they can be used. In some cases the ostium is inaccessible, which would preclude the passage of a cannula. Laminagrams have been of value in a few instances. As for the objection to using a chisel to penetrate the anterior wall, Dr. Maxwell prefers the burr and curette; but in some cases, where the wall is very thick, even the chisel will require persistent hammering to effect an entrance. Like Dr. Bordley's experience, he, too, has found cases referred by the neurologist after having passed through the hands of other rhinologists. Certainly the nasopharyngoscope is a very useful instrument, yet in cases where the sinus is closed off, it will yield no more information than the mirror. In the diabetes cases referred to by Dr. Broyles, it would be difficult to define the etiologic implications except to say that diabetics, being prone to infection, may harbor a long standing sinus infection which has not been diagnosed. Thus the cause and effect relationship may be reversed.

CONCERNING THE CRITERIA OF OPERABILITY IN LARYNGEAL CANCER.

JOHN J. O'KEEFE, M.D.

Dr. O'Keefe discussed the criteria of operability in cancer of the larynx on the basis of an analysis of several hundred cases. Since the objective is the total removal of all of the involved tissues, much depends on the appraisal of the extent and location of the lesion. It was found that only those which were confined to the membranous portion of the cord were

suitable for laryngofissure; however, when the growth has extended to the anterior commissure, the arytenoids or into the ventricle, procedures less radical than total laryngectomy were followed by a high percentage of local recurrences. The figures showed 5.2 per cent recurrence following laryngofissure, 21.4 per cent following anterior commissure technique and 82.3 per cent after partial laryngectomy.

Extra cordal cancers are so frequently associated with cervical metastases frequently undetectable by palpation that failure will frequently follow a total laryngectomy unless a neck dissection is done at the same time. The figures showed 24 per cent of failures after laryngectomy alone and 13.8 per cent after laryngectomy with neck dissection. In cases where cervical nodes were already present, the percentage of failures was 37.5 per cent when neck dissection was not done as compared to 12.5 per cent when it was.

DISCUSSION.

Dr. Louis H. Clerf agreed with the essayist that failures in the surgical management of cancer of the larynx usually resulted from inadequate primary surgery. Recalling Mackenty's observation that the actual size of a cordal lesion was usually one-third greater than the area visualized, he emphasized the point that the resection must be well beyond the apparent lesion if a cure is to be expected. In cases where laryngectomy is indicated, he pointed out that the literature has revealed that 20 to 30 per cent of laryngectomized patients presumably free from cervical metastases develop these lesions postoperatively. In 1955, Dr. Clerf presented a study of the value of block dissection as a primary, a secondary or an elective procedure, and he feels that laryngologists should become more conscious of this trend. Elective block dissection should be considered as part of the operative procedure when laryngectomy is indicated except when the surgeon is convinced that metastasis has not already occurred. The figures presented by Dr. O'Keefe bear this out.

Dr. Julius McCall referred to his movie which was presented 14 years ago, showing combined laryngectomy and neck dissection, which was at that time criticized as being too radical; however, today, Dr. O'Keefe's presentation fully vindicates Dr. McCall's stand. Dr. O'Keefe's survival rate is better than his own, which is 37 per cent. During the past five years at St. Luke's Hospital they have done more neck dissections in combination with laryngectomy than the latter alone. Much discussion nowadays in this connection is concerned with conserving the voice. Dr. McCall has yet to find a dead man who needed a larynx!

Dr. O'Keefe concluded by expressing the hope that further comments might be forthcoming informally.

THE LARYNGEAL SACCULE.

EDWIN N. BROYLES, M.D.

Dr. Broyles studied the laryngeal saccule or ventricle in 50 larynges (100 ventricles) removed at autopsy, 45 per cent from colored and 55 per cent from white individuals. He found twice as many large saccules in the specimens of whites than blacks, and more in males than females. The depth of the saccule varied from 6 to 8 mm. (75 per cent) to 10 mm. or more in 25 per cent, and over 15 mm. in 7 per cent.

These saccules can be demonstrated in tomographs, especially if of fairly large size. Bilateral saccules were twice as common as unilateral.

The importance of examining and studying this anatomical structure cannot be overemphasized, since it may be the seat of undetected pathology. Cell studies, cultures and tomographs may all contribute to the diagnosis of concealed infection of neoplasm.

DISCUSSION.

Dr. Paul Holinger brought up the question of the nature of the sac. Does it have a thin stalk or a broad opening? If the latter, where was the point of demarcation between the saccule and the ventricle itself? Part of this question has been answered by the illustration showing the dye in the injected specimens; but the dye in these appears to fill the entire ventricle rather than merely the saccule. Does the saccule ever have a constricted neck opening into a larger chamber above? Potentially the sac may extend through the thyrohyoid membrane as a laryngocele or posteriorly into the aryepiglottic fold. Has Dr. Broyles been able to find such potentialities?

Dr. Joel J. Pressman told the story of a musician playing a wind instrument whose inflating laryngocele attracted more attention than his musicianship and was referred for surgery. At this time, the father of the boy, an attorney, threatened to sue Dr. Pressman for assault if he operated; however, the young man having just reached his majority was willing, and the operation was performed as per Dr. DeGraaf Woodman's technique. When the larynx was opened, the laryngocele was not found; but when the anesthetist put positive pressure on, it did show up and was removed. Now Dr. Pressman would gladly turn the patient over to Dr. Broyles, who had seen him originally, and was now free to operate the other side if he wished to risk being sued for assault!

Dr. Broyles stated that he believes a saccule of appendix is present in all larynges. It can be differentiated from the ventricle, being merely an extension upward instead of lying just under the false cord. It is usually pyramidal in contour and more frequently in the midline than anterior; rarely posterior. He would be happy to have Dr. Pressman operate the other side since he has already survived one risk of a suit!

PSEUDOSARCOMA OF THE PHARYNX AND LARYNX.

DANIEL C. BAKER, M.D.

Pseudosarcoma of the pharynx and larynx was described by Baker as a non-malignant connective tissue tumor usually associated with squamous cell carcinoma. Although the tissue may grossly resemble a sarcoma by reason of mitotic activity, chromatin pattern and fasciculated bands of connective tissue, the abundance of typical mitoses, and absence of nucleoli in many instances, edema and cellular infiltrate, etc., all speak against such a diagnosis. Investigations by other observers indicate that the lesions are primarily squamous cell carcinoma with the secondary sarcomatous element representing a reactive phenomenon which is non-neoplastic.

Three cases presented by the essayist demonstrate these points.

DISCUSSION.

Dr. Daniel Cunning recalled that Virchow had described this tumor a century ago, showing that it was both a carcinoma and a connective tissue tumor. He called it a carcinosarcoma. Others more recently in this country have demonstrated the same thing. It would appear that the more correct term would be "pseudosarcoma" since it is more often benign than malignant. An awareness of this condition is important for laryngologists, since so many of these growths appear in the respiratory passages. Frequently a mass appearing to be malignant is reported otherwise. Yet it is important that various areas be biopsied before a final diagnosis can be arrived at. The three cases presented by Dr. Baker exhibit various phases of the problem. First, a cancer of the skin and 36 years later a cancer of the throat, all after X-ray therapy in childhood. Then the case of a pedunculated mass obscuring the underlying carcinoma and finally the granuloma of the trachea without any evidence of squamous cell carcinoma.

Dr. Baker had nothing further to add in the discussion.

CYSTIC FIBROSIS OF THE PANCREAS AND NASAL MUCOSA.

MOSES H. LURIE, M.D.

Cystic fibrosis of the pancreas is a disease which involves the mucosa of both the respiratory and alimentary tracts.

Respiratory tract symptoms appear early with repeated colds, wheezing, nasal obstruction, dry cough, bronchitis, bronchopneumonia, bronchiectasis and ear infections. The submucosal glands are involved and secrete a viscid mucopurulent discharge. This disease is accompanied by a high concentration of sodium and potassium chloride in the perspiration, and a simple test devised by Shwachman and Gaha may help to differentiate nonspecific mucosal affections from a true cystic fibrosis of the pancreas. The test consists of placing the patient's hands and fingers on a specially prepared agar plate which has a definite amount of silver nitrate and potassium chromate. A high concentration of chlorides in the sweat will cause a deep discoloration, and the degree of discoloration arbitrarily decided as one, two and three will so indicate. It has been found that in 140 cases of cystic fibrosis, 138 gave a three plus reaction whereas in 77 normal children the reaction was one plus in 65.

These cases should be treated symptomatically and conservatively, avoiding surgery except for removal of polyps.

DISCUSSION.

Dr. Bernard McMahon called attention to the nasal symptoms which often suggest an allergy. Hyperactivity of the glands results in a discharge of mucous, often quite thick. As a result, the ducts of the glands become blocked, and the mucosa herniates forming polyps. Frequently the staphylococcus is found on culture. Sensitivity tests are not always conclusive, but chloramphenicol seems to be the most effective antibiotic. Surgery is not indicated in these children, but it is important that the nasal symptoms be recognized as due to the general process and treated as part of the whole. Dr. McMahon inquired about the use of a 5 per cent cocaine solution mentioned by Dr. Lurie. Did he not think there was a possibility of toxic reactions if the solution were accidentally swallowed.

Dr. Robert E. Priest asked Dr. Lurie whether he had found any serous otitides of the gluey type in fibrocystic disease.

Dr. Daniel C. Baker, Jr., mentioned that he is associated with a New York hospital where Dr. Dorothy Anderson, who described this disease, is the pathologist. The laryngologist is interested in the process because these patients are often referred to him because of the nasal or the broncho-pulmonary symptoms. He has noted the fact that this disease is only one which will produce nasal polyps in a small child. In patients with pulmonary symptoms one must avoid the use of atropine or scopolamine when surgery is performed, because of the tendency to a foreign body reaction which may result in pulmonary insufficiency. This latter has been noted in cases where the disease has affected older age groups—individuals in their 20's.

Dr. Oliver Van Alyea thought that several of the X-ray pictures shown by Dr. Lurie disclosed what appeared to be polyps in the antrum. Would

it not be advisable to remove these since they so frequently are the stalks of recurring nasal polypi?

Dr. Joseph P. Atkins was pleased to hear the rhinological side of the picture as described by Dr. Lurie. He, himself, has been more interested in the pulmonary aspect of the problem. Studies are in progress to determine the composition of the thick secretion which clogs the air passages. In the meantime, mechanical aspiration of these secretions has been of some considerable help.

Dr. Lurie explained that the cocaine solution he has been using is a 1/5 of 1 per cent solution, which being employed off and on has proven very effective and has never caused any outward symptoms. Naturally it is not to be used indiscriminately. As for surgery, Dr. Lurie has found that the disease is not limited to the antrums, and consequently it is often necessary to clear out the ethmoids and often the frontal. Antrums that have been opened for the second time have revealed persisting function of the lining, as evidenced by mucous secretion and goblet cells. Dr. Lurie did have one case in a child where the middle ear contained a very thick gluey mass which, when extracted, retained the shape of the cavity. As Dr. Atkins said, it is not safe to use atropine or scopolamine in these children. Dr. Lurie confessed that he doesn't often see the cases when the pulmonary symptoms predominate, because these are already in the hands of the chest surgeons. It would be well that the chest men be aware of the frequent association with sinus pathology and consult with the rhinologist. Replying to Dr. Van Alyea, he stated that he does remove polypi whenever the diagnosis is certain.

CAUSES OF FAILURE IN SURGICAL TREATMENT OF MALIGNANT TUMORS OF THE LARYNX.

CHARLES M. NORRIS, M.D.

Norris attributes failure following surgery for carcinoma of the larynx to delay in diagnosis, inaccurate appraisal of the extent of the lesion, insufficient biopsy and incomplete removal.

Improvement in percentage of five-year cures is attributed to wider variety of procedures for partial laryngectomy and the recent trend toward more frequent neck dissection. The procedures advocated for invasive lesions of the middle third of the cord, extension into the ventricle and minimal subglottic extension are the fronto-lateral resection with removal of a vertical anterior segment of the thyroid cartilage and an extended form with use of skin graft and mold. Of 209 cases undergoing initial partial laryngectomy the incidence of failures due to recurrence was 7.2 per cent and those due to

metastases 2.4 per cent. Nine of 15 recurrences were salvaged; eight by further operation and one by irradiation.

In 181 cases of laryngectomy without simultaneous neck dissection the incidence of recurrence was 11.6 per cent, and that of cervical node metastasis without recurrence 20.4 per cent.

Postoperative metastasis after laryngectomy without neck dissection, studied in relation to location of the laryngeal lesion, occurred in 6 per cent of the endolaryngeal, 16 per cent of the subglottic, 29 per cent of the vestibular and 42 per cent of the marginal cases. Better results were obtained in 22 cases where the neck dissection was simultaneously performed with the laryngectomy. It is conceded that neck dissection is indicated in most laryngectomies even though palpable cervical nodes are absent.

DISCUSSION.

Dr. F. Johnson Putney explained the finding that all of the patients developing cervical node metastasis after partial laryngectomy failing to recover, as being due to an exceedingly rapidly growing and undetected early metastasis even in the presence of a small lesion. Mobility of the cord is a good criterion on which to base the indications for various procedures, but is sometimes difficult to determine, especially if the arytenoid alone is mobile. Superficial cord lesions with good cord mobility are favorable for irradiation if administered by a roentgenologist who is familiar with larynx pathology. Cancer developing adjacent to the stoma or peritracheal carcinoma represents metastasis to the paratracheal lymph nodes. Cases of associated carcinoma of the lung and larynx are frequently entirely separate lesions. Putney deplores the practice of passing an endotracheal tube for anesthesia through a larynx containing carcinoma because of the danger of implanting carcinoma cells in the deeper structures. He agreed that the prophylactic neck dissection is indicated in all cases except cordal lesions. Bilateral neck dissection, when indicated, should be done in two stages.

Dr. George F. Reed added his approval of neck dissection in all cases involving more than the cordal area. He feels that the expectant attitude in cases where palpable nodes are not found, is an illusory crutch that should be discarded. He will be reporting on an anaylsis of 200 cases from the Massachusetts Eye and Ear Infirmary, showing that in cases where positive nodes were found on neck dissection only 17 per cent survived for three years.

Dr. C. L. Jackson felt that there was too much controversy in the matter of conservative vs. radical surgery. In the words of Professor Sebileau of Paris, surgery shall be "A la demande de lesion." The thing to do is always to be critical of our procedures and seek to benefit by our experience. As Dr. Norris has indicated, the procedures at the Jackson Clinic vary from limited cord resection to laryngopharyngectomy and bilateral neck dissection. The laryngofissure operation they perform is a form of partial laryngectomy, of which there are several varieties. In

some cases the partial laryngectomy is more radical than a simple total laryngectomy. Reports such as Dr. O'Keefe's and Dr. Norris' are valuable because of their analysis of the material from the anatomical viewpoint. Terms such as "intrinsic" and "extrinsic" are obsolete. Another important point is the value of consistently tenacious following up of the cases. It is equally as bad not to hear from patients who have been recovered for more than five years as to fail to be notified of the death of some who did not survive.

Dr. Norris was glad to hear Dr. Putney's warning about the danger of carrying cancer cells into the lower respiratory tract by the passage of a scope through the affected area in the larynx. Dr. Reed's comment that doubtful nodes must be considered malignant rather than otherwise is well taken. He further expressed his appreciation to Dr. Jackson for his suggestion in the matter of classifying these growths.

HISTOCHEMICAL STUDIES OF THE PATHOGENESIS OF NASAL POLYPS.

ALEX WEISSKOFF, M.D.,
and
HELEN F. BURN, M.S.
(By Invitation)

Weisskoff and Burn made a study of polyps removed from 23 patients, 17 being allergic, three infective and three with abnormal mucosa from the antrum, in order to ascertain the nature of the nasal polyp. They found that the histochemical procedure was superior to simple hematoxylin eosin staining, particularly in defining the so-called basement membrane which turned out to be a dense collagenous infiltration with very little of the acid mucopolysaccarides, which are a constant component of the basement membrane.

Histochemical studies revealed the constant presence of acid mucopolysaccaride ground substance in all polypoid tissue, even in the so-called edematous cyst; also, an increased amount of ground substance with increased tissue activity as evidenced by increase in primitive fibroblasts and capillaries. They found evidence that the primitive fibroblast or mesenchymal cell, as well as the endothelial cell, is active in the formation of the ground substance.

The presence of active connective tissue elements, both cellular and stromal, suggests that the polyp is more than

edematous nasal mucosa; it appears to be a connective tissue response to chronic stress of the nasal membranes.

DISCUSSION.

Dr. Walter P. Work commented on the similarity of the ground substance content of both the inflammatory and allergic polyp. Vagaries of the histochemical method can be controlled, to some degree, by using fresh testicular hyaluronidase and umbilical cord in each staining group. These studies show that edema, previously considered to be fluid accumulation, really consists of pools of mucopolysaccarides of varying degrees of polymerization. The concentration of this material in the endothelial cells suggests the possibility of further studies on such lesions as atrophic rhinitis, midline granulomas and similar processes. The relation of these substances to adrenal cortical secretions has been the subject of some investigation in recent years and should provide an area of worthwhile research.

Dr. Harry P. Schenck believes that Dr. Weisskoff's findings will help to confirm the results of electrophoretic studies previously reported, delineating the protein structure of the polyp. While there was no increase in the protein content, they found an increase in the gamma globulin with 100 times the amount of specific antibodies concentrated in extracts of polyp fluid. This procedure is not easy in many cases when the polyps are more solid. As a matter of fact, nasal polyps should be called myxoid fibromas or fibromata; also the more plasma cells that are found, the greater the concentration of gamma globulin. On the other hand, the gamma globulin had no connection with the number of eosinophiles present. Such investigations as the one reported by Dr. Weisskoff should be of great help in clarifying some of these points.

Dr. Weisskoff agreed with Dr. Schenck that in most cases the term "myxoid fibroma" fits the pathologic picture best. The work just reported will be carried on with several other connective tissue histochemical stains and at least a dozen enzymes, which by breaking the findings into various categories, should be fruitful of decisive findings.

WEGENER'S GRANULOMATOSIS.

JOSEPH P. ATKINS, M.D.,
and
SYLVAN EISMAN, M.D.
(By Invitation)

Wegener's granulomatosis is described as a syndrome consisting of necrotizing granulomata of the upper and lower respiratory tract, generalizing necrotizing vasculitis and glomerulitis leading to uremia and death.

It is to be differentiated from lethal midline granuloma in that the latter fails to show the vasculitis which characterizes

Wegener's syndrome, and is unaccompanied by renal involvement. The condition must also be differentiated from Boeck's sarcoid, polyarteritis nodosa and specific granulomas.

Wegener's granulomatosis is classified as being within the group of diffuse collagen diseases. The lesions may involve areas in the upper respiratory tract, the lungs and bronchi and the kidneys, all of which reveal signs of vasculitis of varying degrees. Cutaneous lesions have also been observed.

Seven cases are presented and thoroughly analyzed. Three of these seemed to represent early or non-fatal variants of the syndrome.

DISCUSSION.

Dr. Henry L. Williams expressed his dislike for eponyms, preferring that a syndrome or disease be given a term or name that would give some hint of the pathologic process. As to the authors' attempt to differentiate the so-called "Wegener's granulomatosis" from lethal granuloma on the basis of the former's being associated with lesions in lower respiratory tract, the latter's being restricted to the middle of the face, Williams countered with the statement that such lesions are common in both disorders. As a matter of fact, he pointed out that generalized lesions in the kidneys, lungs, skin and blood vessels are found in a number of diseases, such as verrucous endocarditis with disseminated lupus erythematosis, nonspecific interstitial keratitis and periarteritis nodosa, all being varieties of collagen disorders. In his opinion, all of these conditions, including those under discussion, are fundamentally periarteritis nodosa with unusual prominence in one or more organs. Since the diagnosis of this process is very difficult, pathologists are often led astray. A correct diagnosis requires the ruling out of such disorders as iodism, glanders, tularemia, leprosy, rhinoscleroma, blastomycosis, etc. Even to rule out reticulosarcoma and epithelioma with necrobiotic tendencies takes repeated deep biopsies and study. Midline granuloma is a collagen disease as evidenced by its response to steroid therapy. Cases that have improved following Roentgen therapy would more likely be reticulosarcoma than a true collagen disease; furthermore, cases of lethal granuloma in several instances have been proven to be epitheliomas after thorough study of the biopsies. It is, therefore, impractical and misleading to apply a descriptive term to a condition which may be only one manifestation of a more fundamental process which is as yet not fully understood and is particularly difficult to diagnose.

Dr. Barton related his experience with a case that appeared to be a progressive atrophic rhinitis with saddle nose and inspissated dried mucosa and a granulated appearance to the mucosa, but which in his opinion was a more serious condition for, despite antibiotic and other therapy, the patient died and the autopsy diagnosis was "Wegener's Granuloma." Since the patient had been at the U.C.L.A. Medical Center it was Dr. Barton's hope that Dr. Pressman might have seen her there and could elaborate on the latter stages of the case.

Dr. Pressman stated that he hoped that it would not be assumed that there was any relationship between the patient's being sent to U.C.L.A. and the bad prognosis given at the time!

Dr. Walter B. Hoover thought some of the comments made by Dr. Williams should be emphasized. As long as the etiology is unknown it is best to call all these midline lesions granulomas. Confusion will still exist, as evidenced by three cases he had seen where competent pathologists reported their diagnosis as lethal granuloma, whereas biopsies from areas remote from the ulcerative lesion proved to be lymphoma or lymphosarcoma. Another case with lesions involving the nasal mucosa had partial turbinotomies done to establish an airway, and the tissue was declared to be nonspecific granuloma. After a symptomless interval of two years, the lesions recurred and now involved the external nose and forehead. The biopsies again were indeterminate. X-rays followed by steroid therapy brought about considerable improvement. Thus all one could conclude was that the steroid therapy was of value though the diagnosis was still uncertain.

Dr. Atkins agreed with Dr. Williams that the linking of names to syndromes is undesirable and that descriptive terms are preferable; however, until we have some idea where we are going we have to follow the literature as it has developed. Whether the condition is a periarteritis nodosa or some other disease we do know that we have a condition which may involve both upper and lower respiratory tracts, but is also a disease which involves the entire economy. Dr. Atkins would not disagree with Dr. Hoover since both he and Dr. Williams pointed to the positive diagnosis of a specific condition when taken at a point remote from the ulcerated area.

THE EXPERIMENTAL USE OF HOMOGRAFTS FOR REPAIR OF THE CERVICAL TRACHEA.

JOEL J. PRESSMAN, M.D.

Reporting the results of his experiments on 200 dogs, Dr. Pressman introduced the word "serendipidy" as being applicable in this connection, inasmuch as information that was not anticipated resulted from the experiments, though the original goal sought for was not attained.

Preserved sections of aorta over polyethylene tubing were used in these dogs to supplant excised segments of the trachea. After a lapse of from a few months to three years, the tissues were excised and examined.

The following results were noted:

1. The prolonged indwelling polyethylene tubes brought about a leukoplakia.

2. The aorta retained much of its original structure showing especially the persistence of the elastic fibers despite marked contraction due to fibrosis.

3. Linear shortening of the aortic implant which had united with the trachea increasing the spaces between the rings.

4. Tracheal cartilage adjacent to the anastamosis exhibited calcification and metaplasia into true bone.

DISCUSSION.

Dr. Daniel C. Baker complimented Dr. Pressman on this fine piece of fundamental research. It would appear that an aortic graft could prove to be a satisfactory replacement for an excised trachea, certainly superior to foreign body implants such as nylon, dacron, etc. In his own experience he had never had a case which required total circumferential excision of the trachea. In one instance a traumatically severed trachea was reunited after pulling the lower portion up out of the chest into which it had receded. The idea of mobilizing the lower trachea for resuture to the upper portion has been advocated and practiced by thoracic surgeons. He would like Dr. Pressman to relate whether he had personally had an experience with such procedures.

Dr. John J. Conley commented on Dr. Pressman's reference to serendipidy in connection with his investigation. From the laboratory point of view four items of interest emerge from the investigation, namely, leukoplakia from the pressure of the acrylic, stretching of the residuum of the trachea, retention of elastic tissue in the aortic transplant and metaplasia in the cartilage rings. Although the clinical value for humans is practically nil, the experiment has great value for the positive facts elicited. Survival of homografts varies according to the tissue involved, the cornea and cartilage heading the list. Dr. Pressman's studies revealed the fact that the elastic fibers of the transplant survived above all the other components; yet, replacement of large segments of the trachea is still an unsolved problem, although defects up to 3 cm. may be closed by approximation. To ease the stretching of the trachea one can "accordionize" it by cutting between the rings. Larger defects can be closed by free tracheal implant which is a composite graft taken from the lower trachea. Dr. Conley then described processes involving the use of homologous materials and his procedure for correcting major defects.

Dr. Alexander Weisskoff was deeply interested in the slides showing the elastic fibers and the conflict between the tissues surviving and those succumbing to fibrous connective tissue inroads. A chronological sequence of slides could afford exceedingly valuable information on the basic understanding of connective tissue. Recently a connective tissue fiber found in the Achilles tendon, and another between the tooth root and surrounding connective tissue have been described. Special stains in Dr. Pressman's series might bring out some new information, particularly with reference to the bony metaplasia he observed.

Dr. Pressman stated that although he had avoided reference to the clinical application of his work, the possibilities are nevertheless in the background. Certainly, as pointed out by Dr. Conley, partial defects of the trachea can be effectively closed by aorta transplant as long as the defect is not circumferential. Such a covering supplies not only support but also an epithelial lining, and prevents occlusion by ingrowing granulations. A point was mentioned about the experimental dogs. Those with aortic implants did not die; they were sacrificed at will. The dogs that carried only polyethylene tubes survived as long as the granulations were taken care of.

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Meeting:

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Meeting:

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Secretary of Ophthalmology Section: Dr. Philip D. Shanedling.
Chairman of Otolaryngology Section: Dr. Robert W. Godwin.
Secretary of Otolaryngology Section: Dr. Francis O'N. Morris.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd., Los Angeles, Calif.
Time: 6:30 P.M. last Monday of each month from September to June, inclusive—Otolaryngology Section. 6:30, first Thursday of each month from September to June, inclusive—Ophthalmology Section.

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Meeting:

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Meeting: Second Tuesday in each month at 8:00 P.M. at Memphis Eye, Nose and Throat Hospital.

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Meeting:

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Meeting:

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Executive Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa., U. S. A.
Meeting: Seventh Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
Time and Place: Miami, Fla., March, 1960.

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